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VETERINARY MEDICINE

FEBRUARY 1938

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Chicago, U. S. A.

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The XIII International Veterinary Congress

As all readers know, the Thirteenth International Veterinary Congress will be held in Zurich and Interlaken, Switzerland, August 20-27, 1938. It is not necessary to tell anyone who attended the Twelfth International Veterinary Congress in New York City in 1934 that these quadrennial or less often international veterinary congresses bring together the leaders and outstanding figures in every branch of veterinary medicine the world over, nor that the program comprises a report of the latest developments in scientific veterinary medicine in all countries; nor to stress the attractive features of these meetings, nor the importance of attending them.

About 60 American veterinarians and their wives attended the Tenth International Veterinary Congress in 1914 and nearly double this number the eleventh congress in London in 1930. The attendance at the New York congress in 1934 exceeded by 100% the attendance at any other veterinary gathering ever held on this continent, and it seems certain that a large number of American veterinarians will attend the thirteenth session of this organization next August.

Two tours of American veterinarians are being promoted to attend this congress, and incidentally visit many of the most attractive places in Europe from a veterinary, scientific and historical point of view.

One of these tours is under the management of Treasure Tours, Inc., 24 West 40th St., New York City. This tour includes in its itinerary, 10 foreign countries and 18 cities. It requires 65 days, July 13th to September 15th. It will leave from New York and return to New York. The total distance traveled from New York to New York is 10,367 miles. The price, $794.00, includes travel, hotels, meals, gratituities and sightseeing—all necessary expenses, except the cost of passports and certain small amounts in taxes.

The director is Dr. Adolph Eichhorn, who directed the American veterinary tours both in 1914 and in 1930, and who has conducted other organized tours of Europe.

The countries visited include England, Belgium, Holland, Germany, Hungary, Austria, Italy, Switzerland and France, and the cities Southampton, London, Brussels, Scheveningen, Berlin, Leipzig, Dresden, Munich, Budapest, Vienna, Venice, Milan, Zurich, Interlaken, Geneva, Grenoble, Nice and Paris.

The other tour is under the management

of Thos. Cook & Son, the oldest and largest of tourist agencies with offices in all the principal cities of the world, more than 350 altogether. The Cook tour includes 14 countries and 23 cities and comprises 55 days, July 23rd to September 15th. It will leave from Montreal and return to New York, traversing a distance of 12,238 miles. The price (all necessary expenses except passports and certain taxes) is $614.00—five cents per mile.

The director of this party is Dr. D. M. Campbell of Chicago, who has had extensive experience in conducting all expense tours.

The countries visited include Canada, Scotland, England, Denmark, Germany, Holland, Belgium, France, Switzerland, Italy, Yugoslavia, Greece, Algiers and Portugal, and in addition Sicily, Gibraltar and the Azores.

The cities visited include: Montreal, Glasgow, Stirling, Edinburgh, London, Copenhagen, Berlin, Hanover, Amsterdam, Brussels, Paris, Zurich, Interlaken, Milan, Trieste, Ragusa, Patras, Naples, Palermo, Algiers, Gibraltar, Lisbon and Ponta del Gada.

Warburton's Old Letter Comes to Light Again

Dr. E. F. Jarrel, Tyler, Texas, reported, in connection with the unemployment census, that county agents and vocational agricultural teachers in his state are engaging in veterinary practice, as, of course, every informed person knows, they are doing in most states.

Doctor Jarrel received this reply:

UNITED STATES DEPT. OF AGRICULTURE
Director of Extension Work
Washington, D. C.
December 10, 1937.

Dr. E. F. Jarrel, Tyler, Texas.
Dear Dr. Jarrel:

Your letter of November 20 to the President has been referred to me for reply. If you will inform me about specific instances where county agricultural agents are performing veterinary services for farmers I assure you I will use my best efforts to correct the situation. We do not want extension agents to compete with veterinarians and frankly I think there are very few cases where this is happening. County agents everywhere are very busy on other duties, and most of them recognize they are not qualified to treat sick livestock.

Yours very truly,
(Signed) C. W. WARBURTON,
Director of Extension Work.

Accepting this letter at what it appears to say, Doctor Jarrel asks veterinarians to supply him with specific instances—approximate dates, places and veterinary work done by county agents and vocational agricultural teachers. This, he will report, together with information of his own, to Mr. Warburton.

If we may in any degree judge the future by the past, Doctor Jarrel will then receive Mr. Warburton's much used second letter to the effect that the Federal Extension Service is unable to control the action of county agents directly, for the reason that this matter is within the jurisdiction of the various state directors of agricultural extension and the Federal department can merely advise them as to what type of work county agents should engage in.

Doctor Jarrel, if he follows the line pursued by others, will then write Mr. Warburton that the congressional appropriation for the support of agricultural extension limits the expenditure of funds solely to educational purposes and will ask Mr. Warburton if he is willing to withhold Federal allotments from states in which it can be shown that county agents and teachers of vocational agriculture are generally engaging in veterinary practice in violation of the laws of those states.

Then will come Mr. Warburton's famous third letter, revealing his attitude in this matter in contrast to his first and second letters, which never reveal it. In a round about way, this letter will tell Doctor Jarrel that it is not the policy of the Federal Extension Service to attempt to control in detail how the Federal funds are expended, but merely that broad policies are outlined and the funds allotted; that it has not been the policy to follow the matter up to see just how the funds are used; that the Federal department feels that the state directors are in a much better position to judge the needs of agriculture in their own states than are officials in Washington. This closes the matter; or has in the past.

Sulfanilamide

By L. A. MERILLAT, Chicago

As you are aware a new internal germicide, officially known as sulfanilamide, has come into the practice of medicine. It was introduced as such by Gerald Domagk of Germany in 1935 when on February 15 of that year he published an article on a remarkable chemotherapy of streptococcic infections in *Deutsche medicin Wochenschrift*. The remedy, a dark red dye of complex formula named prontosil, was synthetized from coal tar by the chemical house of Mietzsh and Klarer. Later, several formulas of the same general character were introduced under various names, among which are "prontylin," "chrysoidine," "di-sulphanilamide," and the more simplified formula "sulfanilamide," the subject of this paper. In simplifying the chemical formula without diminishing the germicide action, the result was a white crystaline powder which does not have the physical properties of a dye in the strict sense of the word. This explains why the product with which you are becoming familiar does not have the staining properties of the dyes, now extensively used in the treatment of disease, of which methylene blue, trypanblue, diamine, acriflavine, gentian violet and pyrrol blue are well known examples.

The discovery of the remarkable germicide action of dyes within the living body, which has occurred during recent years, represents the greatest achievement of chemotherapy since the natives of Peru more than 400 years ago discovered the curative properties of quinine in malaria and Ehrlich, at the turn of the nineteenth century, demonstrated the germ-killing power of colloidal arsenic in the treatment of syphilis. Trypanblue and diamine are specific cures for surra, a trypanomiasis of the tropics. Methylene blue has a pronounced detoxicating action against prussic acid and certain catabolic toxemias. Gentian violet, as you know, attenuates the virus of hog cholera without destroying its antigenic properties. Pyrrol blue given intravenously and locally dissolves neoplastic tissue. To these specifics, one now adds sulfanilamide, which has proved of value in the treatment of streptococcic, pneumococcic, gonococcic and meningococcic infections and is so evaluated in modern medical science.

Considerable change in the evaluation of this chemical has occurred since January of this year. In the January second issue of the *Journal of the American Medical Association*, sulfanilamide was said to be useful only in the treatment of certain hemolytic strains of streptococci. Since that time, however, investigators in this country and abroad have demonstrated its germicidal action in a variety of microbic infections, among which are scarlet fever, erysipelas, puerperal metritis, pneumonia, types I, II and III, infected abortion, septic sore throat, and contaminated traumatic wounds.

The list of diseases of animals for which sulfanilamide may be scientifically employed remains to be made up. The happy prospect is based upon the fact that its therapeutic value was determined by animal experimentation, and that clinical reports gathered up to the present time are promising. But as in all innovations which come into medicine from time to time one should avoid over-enthusiasm until time and usage has set the actual values. It is a close relative of a large number of well known chemical compounds, which are highly poisonous, and while sulfanilamide does not have the prompt lethal effects of most of its relatives, its possible capacity for destroying life should be respected until its whole effect is definitely known.

There is still much to be learned about the dosage of sulfanilamide. It requires a certain amount of concentration in the body to overcome a certain degree of infection. This seems to be quite mathematical so far as human and experimental medicine is concerned. In our field, we still

*Discussion at a quarterly meeting of the McClean County Veterinary Medical Association, Bloomington, Ill., Sept. 26, 1937.

lack definite direction for dosing, for example, a cow gravely stricken with mastitis, or obstetrical metritis, or a horse suffering from grave pleuropneumonia. This necessary information will come only with time and the experiences of practitioners. As a whole, the results so far reported need further corroboration. Our recommendations are based upon observations made in horse markets on horses affected seriously with the complications of influenza. Much work on dosage remains to be done in cattle and dogs. That is, the amount required to effect the necessary concentrations of the chemical in the blood of the patient remains to be determined, as is also the duration of the action of this new drug. What we know in this respect is based upon the findings in human medicine, where according to Marshall, Emerson and Cutting (*Jnl. Amer. Med. Assn.,* March 20, 1937) a concentration of from 1:5,000 to 1:10,000 can be attained and maintained in the blood. The determination of the concentration by analysis is said to be essential. Besides it seems that this concentration must be maintained for several days to accomplish the purpose. Since veterinary fees are too small to justify making the delicate tests required to determine the concentration, we shall have to be content with decisions coming from the field of human medicine.

The authors above mentioned found that a quick concentration can be attained with a large single dose of 0.05 gram per kilo of body weight. This would mean 25 grams or nearly an ounce for a 1,000-pound horse, and this requires to be repeated every four hours. The maximum quantity that may be administered in man is 4.5 grams, or slightly over a dram. Since we have no information to the effect that 25 grams would be tolerated by a horse or cow of 1,000 pounds weight, or if dogs would tolerate the equivalent, our tests and clinical experiences have led us to make certain recommendations on dosage which we pass to practitioners with an open mind ever ready to make the changes which additional experience may teach.

We recommend that a 1,000-pound horse should receive 90 to 120 grains the first day,

L. A. Merillat

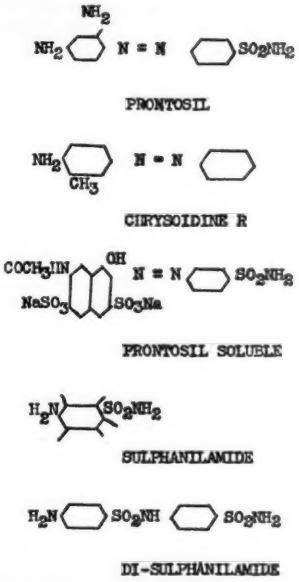
a slightly lower amount the second day, and still less on the third and fourth days. This, we believe, will maintain a concentration capable of exerting the desired microbicide action. For dogs, we recommend 40 grains the first day in four doses four hours apart and 20 grains on the subsequent three days.

The question of intravenous medication with sulfanilamide is constantly cropping up. In this connection, there is but one answer. Don't do it, just now. The chemical passes quickly unchanged into the blood from the alimentary canal and the desired concentration can be attained and maintained better from oral dosing.

Because the absorption in the alimentary canal is complete in four hours, dosing at intervals of four hours has become standard for the first day of heavy dosing and thereafter it is said that, once the concentration of 1:10,000 has been reached, tthe exact amount given thereafter can all be found in the urine. If the amount found in the urine is less than the amount given orally, the rest has been retained to maintain the original concentration, like water overflowing from an already filled vessel. Perhaps this is quite theoretical, but it tends to emphasize the doctor's objective in prescribing this chemical. That is, the idea is to keep the body loaded until the infection is overcome.

In dogs, the substance is excreted in the urine quite unchanged, while in some laboratory animals some of it is conju-

gated. This being true, urinalysis would not be an exact guide to subsequent dosage. Anyhow, the main idea is to try to establish the equilibrium between the intake and



Graphic Formulae of the Aminobenzenesulphonamides*

the elimination. In carefully made tests in man, it was found to require two to three days to establish that equilibrium (Marshall, Emerson and Cutting *supra.*). The fact that sulfanilamide can be found in the cerebrospinal fluid of dosed patients, points to its value in nerve-center infections such as one encounters in canine distemper and equine infectious encephalomyelitis. It should be mentioned, however, it is not known to have any value against filterable viruses but only against certain members of the cocci family, and in the foregoing can be thought of at present, only in connection with secondary invaders.

In conclusion, one should confess that the method by which sulfanilamide inhibits microbian growth within the living body is not known. The popular idea that it is bacteriostatic has not been proved and no other theory is even that acceptable.

*Prepared by Ashe Lockhart from *Public Health Reports.*

EDITOR'S NOTE.—All veterinarians of course know of the disaster—death of 73 persons—from "elixir sulfanilamide" placed on the market untried by Messengill of Bristol, Tenn., and Kansas City. Regrettably the account of this catastrophe in *Time* magazine and many newspapers stated that Messengill is a manufacturer of veterinary remedies. That may have left in the minds of some the impression that veterinary medicines are placed on the market without testing or their toxicity being determined. Of course, such is never the case among reputable pharmaceutical houses catering to professional veterinarians.

It should be known that Messengill is a manufacturer of animal medicine sold direct to the owners of animals and that his veterinary customers include only that small fraction of veterinarians whose criterion for the selection of medicaments is price alone. After all why should anyone be surprised that a purveyor of potent drugs for lay use should place on the market untested preparations? What is the use of supplying a fine product to those who do not know how to use it? If a manufacturer is so indifferent to the verities as to call diethylene glycol, a near relative of prestone, an elixir (officially an elixir is a sweetened, aromatic, alcoholic liquid) why should anyone be surprised that he should be unconcerned as to the effect of the excipient?

A Disastrous Experience with Animal Disease*

I am sure that you as veterinarians realize, and I wish the entire population of this state could realize what a priceless heritage Wisconsin has in the advancement of its animal disease control program. I think no one in the state has a better chance to realize how priceless this heritage is than myself, because I once lost 22 animals on a single test in my own pure-bred Guernsey herd from tuberculosis infection. I had Bang's disease in my herd continuously from 1891 until October, 1934. The losses in my own herd from these two diseases are almost beyond comprehension of anyone who has not lived with the problem from year to year.

There is another thing that I wish the farmers of Wisconsin might more fully realize, and that is the debt of gratitude they owe to the veterinary profession for the part it has taken in these disease eradication campaigns.

*From an address by Chas. L. Hill, Commissioner of Agriculture and Markets of the State of Wisconsin at an Annual Meeting of the Wisconsin Veterinary Medical Association, Jan. 19, 1937.

Reasons Why E. B. McLean Race Horses Headed the List of Money Winners in 1928[1]

By ROBERT LEE HUMPHREY,[2]
Mountville, Va.

July 1, 1926, at the invitation of the owner of Belmont Plantations, Leesburg, Virginia, I took charge of his farm, which was devoted entirely to breeding race horses, for a two-year period. These horses were raced by their owner and breeder who raced no others. Although he had spent considerable sums of money to secure good breeding stock and no expense was spared in training these horses, the results had been disappointing. For the ten-year period ending in 1926, the stable had won an annual average of $36,364. While this figure may seem fairly substantial, it was not in keeping with the number of horses bred and trained.

The owner was inclined to put a large share of the blame on his two stallions, which had been extensively used. Colin had been unbeaten as a race horse, winning the most important stakes of his day, but as a stallion was generally regarded as a pronounced failure. The owner warned me that the other stallion, The Porter, was a failure also.

The trainer added that all the colts by The Porter broke down in training before they could be got ready in a thorough way for racing. They suffered, he said, from bad legs and soft joints.

After hearing a good deal about how bad the horses were, I went to the farm for a thorough inspection. At first glance it was apparent that this farm was lacking in sufficient grass to carry the number of horses quartered there. However, on the whole, the physical condition of the colts looked satisfactory. Their coats were bright and they were fat.

Looking for a clue as to where to start, I took blood samples from various colts—the produce of a cross section of the mares, that is, from maiden mares, from mares having had a few foals and from old mares having had many foals. These blood samples were sent to a laboratory (the biochemical laboratories at Johns Hopkins University) for analysis. The report came back that all these animals, without exception, were badly deficient in calcium and phosphorus. Here was a clue worth following, pointing to a condition which could be remedied.

Fortunately there was a considerable accumulation of manure in the cattle yard. This was spread at once, and followed by an application of one ton of ground, burnt lime to the acre. After the first rain, the land so treated was re-seeded with mixed clovers and a small amount of blue grass. Following this, on the same land, 350 pounds of 16% super-phosphate to the acre was spread.[3] These applications were made at the far end of the colt and filly pastures, partly because that end of the pastures had been badly neglected, and partly because the best grass there would mean additional exercise for the stock going to and from the barns. However, so attached did the colts and fillies become to these far ends of their paddocks that it was often necessary to send a man down to bring them back, even at feed time. Their preference for these fertilized and

[1] An address before the 18th Annual Meeting of Members of the Horse and Mule Association of America, Chicago, December 1, 1937.

[2] A year ago, an address at the 17th Annual Meeting of the Horse and Mule Association of America on "Lameness in Horses—Its Relation to Their Feed", by Dr. D. M. Campbell, attracted world-wide attention. This address, by Dr. Robert L. Humphrey, is a follow-up to Dr. Campbell's discussion. It would not have been available but for the alert attention of Abram S. Hewitt of Montana Hall, White Post, Virginia, a breeder of Thoroughbreds, who advised me that Dr. Humphrey had facts bearing on the subject. Doctor Humphrey was interviewed, and while reluctant to make an address, he finally agreed to prepare a statement answering questions propounded. Especial thanks are due Mr. Hewitt for his valued assistance, and to J. A. Estes, Editor of the Blood Horse, Lexington, Kentucky, and R. J. Brooks, Editor of Daily Racing Form, 441 Plymouth Court, Chicago, Illinois, who placed important records of their offices at our service.—Wayne Dinsmore, Secretary, Horse and Mule Association of America.

[3] Ordinarily, manure and lime are spread in late fall and winter months and super-phosphate in March: but the necessity of prompt treatment led to immediate use of manure and lime in August, 1926, and super-phosphate early in September, 1926. Fortunately fall rains came, making the fertilizers promptly available. Long continued tests by State Experiment Stations, however, indicate that winter and spring application—the usual system—is best, for fall rains do not always come when needed.—Wayne Dinsmore, Secretary, Horse and Mule Association of America.

mineralized plots, which they had previously scorned, was unmistakable.

All the horse manure from the barns was run through the cattle yard, and tramped down by 40 head of cattle kept for that purpose. The cattle were fed cottonseed meal, shelled corn, ensilage and hay. Hogs were kept with the cattle in order to work up the manure, add their own manure to the mixture, and eat the grain undigested by the cattle. In working up this manure, the hogs exposed a great deal of it to the air and sunlight, thereby helping to exterminate the parasites in the horse droppings.

As manure became available from the cattle yard, it was spread on the horse pastures.

In addition, the feeding schedule was changed. The cook room was closed, and during my time at the farm was used only as a store room. No feed was cooked. Still following the clue of mineral deficiency, the heaviest oats obtainable were brought from the Pacific Coast at a cost of $1.00 per bushel, delivered at the farm. The mineral content of these oats is so heavy in comparison with standard oats that for purposes of supplying mineral deficiency they appeared cheap even at that price. With these oats was fed green alfalfa meal not exceeding one pint per feed together with bran, yellow corn and linseed meal. The colts were also fed a cod-liver-oil emulsion containing calcium and phosphorus. This emulsion was fed for 10 days, then suspended for 10 days, then fed for another 10 days. During the 10-day suspension, mineral oil was substituted. This was all the grain ration, based on the old theory that hard grain makes hard horses.

At the end of 90 days from the time this feeding schedule and mineralizing program had been put into effect, blood samples were again taken from the same animals as before. This time, instead of reporting by telephone, the bio-chemist from Johns Hopkins Laboratories called personally to find out what had made such a radical increase in the quantity of minerals showing in his analysis of the blood samples.

At this point I should explain that I followed the course I have just outlined because I believed that it is better to supply these minerals through the natural feeds as much as possible, rather than through artificial concentrates.

The horses which had been racing for the stable in 1926 came back to the farm late in the fall of 1926 for the winter of 1926-27. They, too, were put on a feeding schedule assuring an ample supply of essential minerals. Naturally it was with some anxiety and a lively degree of curiosity that I waited to see what the 1927 racing season would hold for our horses.

The results were emphatic and surpassed my rosiest hopes. In 1926 the stable had won about $46,000; that is, about $10,000 more than its ten-year average. In 1927 it won over $186,000. In 1926 it won 28 races; in 1927 it won 58. The number of races won doubled, but the amount of money won, quadrupled.

The year 1928 was even better. The stable won 60 races worth $234,640, putting the owner in the enviable position of leading the list of money winners.

On July 1, 1928, my contract came to an end, and I left the farm. I felt that the yearling crop that year was much the best so far reared at Belmont. Unfortunately these horses were taken South for winter racing. Practically all of them contracted influenza of a very severe type. Many of them were raced perhaps too soon after this sickness. In any event, the horses that I was confident would prove the best group sent into the racing stable from the farm, turned out to be of very little use for racing.

After I left Belmont, the practices I had adopted to assure an ample supply of minerals, was not continued. It is interesting and perhaps significant that the winnings of the stable fell rather sharply to a point about the same as where they had been before the mineralizing program had been put into effect.

In this story there is one point of cardinal interest. The owner in question raced only the animals he had bred himself, with but one exception that I can recall. The

large winnings, therefore, were entirely made, to all intents and purposes, by animals bred at Belmont. When given an adequate supply of minerals contained in a proper feeding schedule these animals proved themselves about the best race horses in the country. Without proper nutrition, they were indifferent performers even when they stayed sound enough to race.

I give this story in some detail because I know of no other case where all the horses of a large racing stable came from one farm,—a farm where the breeding stock remained the same both before and after the experiment in nutrition,—and where the horses came from comparative obscurity to the pinnacle of success and then after the experiment in nutrition was discontinued, returned to obscurity.

* * * *

The Veterinarian and His Microscope

The progress made by applied parasitology and its importance in the raising of animals has been made possible through the use of a microscope. Every veterinarian should possess a good microscope, either a reconditioned or a new one. A microscope in a veterinarian's possession impresses his clients favorably. Furthermore, the public demands correct diagnosis and treatment of ecto- and endoparasitisms, and without the aid of the microscope this is utterly impracticable if not wholly impossible in many cases.

Many owners of horses, cattle, cats, dogs, foxes, etc., know that the presence of internal parasites may be detected by fecal examination with a microscope. Specific anthelmintic treatment may thus be administered for parasites.

Some of the parasites eggs that may be found and identified by fecal examination are: tapeworms, hookworms, strongyles, ascarids, whipworms, pinworms, and many others. A positive diagnosis of coccidiosis can be made by fecal examination, or by scrapings from the rectal mucosa. Hematoidin crystals found in the feces are of value to the veterinarian for the diagnosis and the treatment of the animal.

What could be more satisfying to the clients than to have his veterinarian show him under a microscope a deep skin scraping from his animal revealing sarcoptes, psoroptes or demodex mange mites? The clients seeing the parasites realize what some of the problems of a veterinarian are and appreciate his efforts in treatment. Other skin scrapings may show various fungi such as ringworm (*Trichophyton tonsurans*), favus (*Achorion schönleinii*) or scabies of fowls (*Cnemidocoptes mutans*).

The positive diagnosis of heart worm (*Dirofilaria immitis*) is largely dependent on finding the microfilaria in a blood smear under a microscope. Leucemia may be differentiated from Hodgkin's disease, or pseudoleucemia, and tuberculosis by the increase in the number of white blood cells in the blood. The Malaria-like disease of ducks due to infection by *Leucocytozoon anatis* may be determined by finding the different stages of the parasite in the blood of the affected bird. Texas fever is diagnosed by finding the *Piroplasma bigeminam* in the blood with the aid of the microscope. Anaplasmosis likewise is diagnosed by finding a small mass of material in the erythrocytes.

In a stained smear of fresh milk one looks for streptococci and leucocytes for help in the diagnosis of mastitis. This may be of great value in locating diseased herds by examination of marketed milk and is widely used for this purpose in milk control laboratories.

In artificial insemination or in natural copulation, the microscopic examination of semen shows the number and activity of spermatozoa which are present. This information is valuable in the selection of males for breeding purposes.

The use of the microscope is one of the means of keeping quackery from intruding upon the practise of veterinary medicine—it marks the scientific man.

FRANK A. CASPER, K.S.C. '38.
Manhattan, Kans.

The Blood Pictures in Two Cases of Infectious Eguine Encephalomyelitis

By M. J. GREGG, K. S. C. '38,
Manhattan, Kansas

September 22 and 23, 1937, two black yearling fillies, affected with encephalomyelitis, were brought to the veterinary clinic of the Kansas State College The first one, designated as Case No. 23 on the clinic records, ended fatally, September 28th. The other, Case No. 20, recovered and was discharged October 2nd. These two cases are described because they represent blood pictures of what may be expected of a recovery and a fatality.

Upon entrance to the hospital neither of the two cases showed severe clinical symptoms. Neither became so weak that aid was required in standing, as is necessary in the large majority of the cases of infectious encephalomyelitis. However, on one or two occasions a nudge or a shorp word was required to get No. 23 to arise.

Case No. 23 seemed to improve greatly the first 24 hours; but on the third day began to show general nervous symptoms and the usual paralysis of the deglutory apparatus. On the sixth day she died quite suddenly a few minutes following the venapuncture. After a post-mortem examination death was attributed to shock and foreign-body pneumonia.

Case No. 20 at no time showed evidence of deglutory paralysis or serious locomotory weakness.

Treatment

Case No. 23 was given 250cc of normal mule serum, 250cc of a 5% solution of dextrose and lgm of proflavine intravenously on the day she was admitted to the hospital (Sept. 22).

No treatment was administered the following day, but on the 24th (third day), 40cc of influenza serum and 150cc of a 5% glucose solution was given intravenously. No further medical treatment was given, but water was supplied daily by means of a stomach tube and pump.

Case No. 20 was given 125cc of a 50% glucose solution intravenously on the day she entered the hospital and 500cc of a 5% dextrose in physiological saline solution intravenously the following day. No further medical treatment was given during the remainder of the stay in the hospital, and she was able to drink as needed.

Blood Examination

Daily venapunctures were performed on these, as on all animals entering the clinic with encephalomyelitis. Erythrocyte, leucocyte and differential counts were determined along with hemaglobin percentages. After the daily data had been recorded the Shilling index was calculated. The following technique was used to determine the results:

For the red and white counts, the standard graduated red and white cell pipettes were used and counted in the Neubauer hemacytometer. The hemaglobin determinations were made with the Haden-Hausser hemoglobinometer. A total of 100 leucocytes were counted on each slide and on this basis the daily differential percentages were calculated.

Although No. 23 entered the hospital on September 22nd no blood statistics were calculated until the following day, which accounts for the absence of the data from the above table on that date. By immature forms is meant the myelocytes, juveniles, and stabs while the mature forms are segmenters or polymorphonuclear leucocytes.

Shilling Index

The Shilling index is the relationship of the immature leucocytes to the total leucocyte count and is recorded in the following graphs on a percentage basis. Shilling has proved that in the presence of infection there is a definite change in the proportions of these cells. In the beginning, due to the toxic impression upon the bone marrow, there is an interruption in the development from stabs to segments with a consequent increase in stabs. This increase in stabs, or immature forms, is known as the Shilling index shift to the left, and in serious infections may be accompanied by a simul-

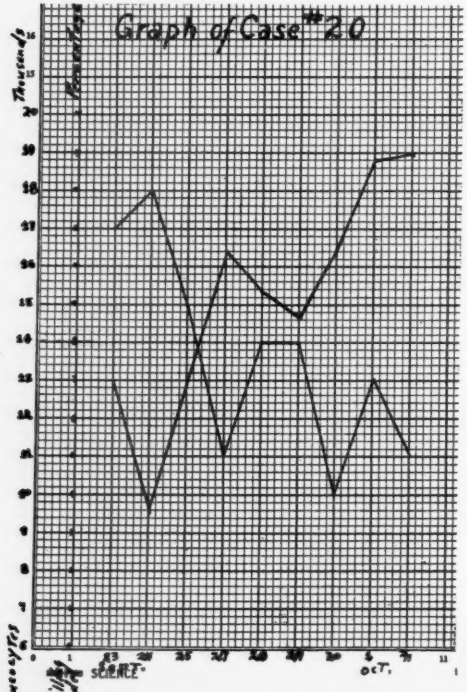
taneous drop in the total leucocyte count. Attention is also directed to the change in number of the lymphocytes during an infection. With a beginning shift to the left, a peak as is indicated by the increased percentage of the immature leucocytes to the highest level, and the lowering of the total leucocyte count to the lowest level Septem-

The following is the daily record of each case:

Date 1937	Temp.	Pulse Rate	Resp.	Eosinophiles	Immature Forms	Mature Forms	Lymphocytes	Monocytes	Total Leucocytes	Total Erythrocytes	Hemaglobin
				CASE NO. 20							
9/23	101.8	44	48	2	11	64	22	1	13,000	11,375,000	11
9/24	102.4	48	30	1	12	53	34	0	9,550	9,637,500	12
9/25	101.7	42	34	2	9	68	21	1	13,000	6,450,000	9
9/27	101.2	42	30	0	5	68	27	0	16,400	9,200,000	12
9/28	101.5	40	32	5	8	62	25	0	15,350	8,562,500	9
9/29	102.4	36	24	8	8	46	38	0	14,700	7,012,500	10
9/30	100.2	36	22	5	4	71	20	0	16,450	7,487,500	12
10/1	101.2	32	24	1	7	56	36	0	18,800	7,325,000	11
10/2	102.0	30	28	3	5	55	37	0	18,950	7,875,000	11
				CASE NO. 23							
9/23	102.0	56	52	1	7	35	56	1	9,500	8,910,000	13
9/24	101.2	54	24	0	3	52	44	1	7,950	6,662,500	13
9/25	102.0	54	20	2	7	44	47	0	7,150	8,687,500	13
9/27	103.0	56	24	0	4	59	37	0	13,350	5,537,500	16
9/28	106.1	138	76	0	13	56	29	2	6,250	12,387,500	18

there is a coincident decline in lymphocytes.

By following the graphic recording closely it will be seen that the disease reached

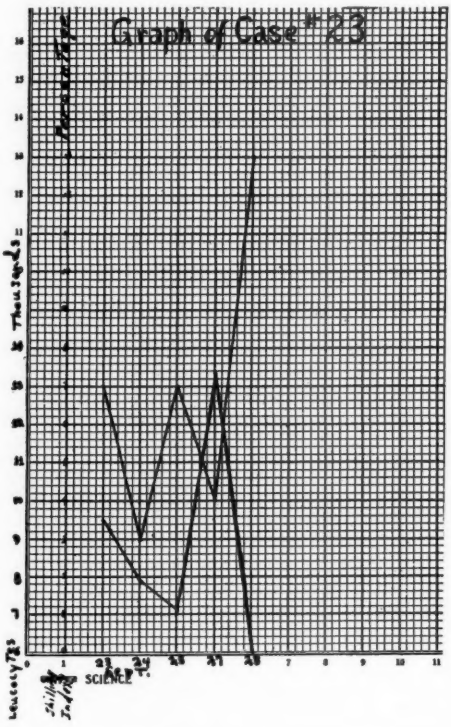


ber 24th. This was true clinically also as at this time the animal showed increased nervousness and irritability, which is also indicated by an increase in body temperature.

On the following day, September 25, the blood picture took a reverse which would indicate improved health, however this was not true clinically. No apparent improvement could be seen until September 27, at which time the clinical symptoms became more favorable with reduced nervousness and an increased desire to eat and drink. By looking at the graph it may be seen that the blood picture indicated improvement at this time as the leucocyte count continued to rise and the Shilling index decreased.

On the 28th and 29th the graph indicates a relapse but there was no clinical evidence of such a setback and improvement seemed continuous throughout the remainder of the stay in the hospital. The continued increase of leucocytes to 18,000 upwards can probably be accounted for as the animal was in a convalescent stage of disease and an increase in leucocytes was necessary to combat the infection. The lowered percentage of immature forms would further tend to indicate that the leucocytes were not being produced abnormally, implying that the

toxic impression on bone marrow is lessened, thus allowing the production of a higher percentage of mature forms.



Case No. 23 presented a somewhat more varied and erratic picture. As stated before, the patient seemed to improve clinically from the time of entrance on the afternoon of September 22 until September 24. Graphically one might conclude that the decrease in percentage of immature forms and the increase in percentage of lymphocytes indicates a retardation of the Shilling index shift to the left, which would point to some slight improvement. It was not until the third day after entrance to the clinic (Sept. 22), that any noticeable clinical change for the worse became visible. It was noticed on the afternoon of the 25th that the animal had become more nervous, was weaving from side to side with the head lowered, and was showing paralysis of the muscles of mastication and deglutition. This is indicated graphically by the lowering of the total leucocyte count and the increase in immature forms on the 25th. However, on September 27 the blood picture pointed toward general improvement but clinically no change for the better was visible. Although able to arise under its own power, with a little urging, the rise in body temperature indicated anything but improvement. On the 28th the blood picture conformed very closely to the clinical picture. The marked increase in immature forms and decrease in total leucocytes indicated a definite relapse. This was true clinically also, as is shown by the marked increase in body temperature, increase in pulse and respiration rates, increased nervousness, general weakness, and death.

Discussion

1. It can safely be said that in these cases the rise and fall in numbers of immature leucocytic forms compared with the total lecocyte counts gives a graphic recording that conforms quite favorably with the clinical picture.

2. On the other hand, on at least four occasions they do not coincide as accurately as could be desired. This may indicate that at least some variances may take place in the blood picture without appreciable clinical changes.

3. It may be noticed that the expected decrease in lymphocytes did not, on several occasions, accompany the increase of immature forms, which would indicate some unaccounted for irregularity in accordance with Shilling's findings.

4. Although the treatment might possibly have altered the blood picture there is hardly sufficient evidence to show definitely that such was true.

5. Since the cause of death in case No. 23 was attributed, on post-mortem examination, to shock and foreign body pneumonia there may be some question as to whether or not the findings in this case represent true infectious equine encephalomyelitis.

6. It should be remembered that the statistics presented represent only two clinical cases and are therefore insufficient upon which to draw conclusions.

Diseases of Canary Birds

By HYMAN POGORELSKY,
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Introduction

OF the various birds kept for household pets the canary bird is by far the most common and the best known. They have been domesticated for centuries. In the United States there are scores of canary bird societies, clubs, and associations. In addition there are several periodicals devoted exclusively to these birds. The total investment in the breeding, rearing, and distribution of canary birds amounts to several million dollars. It is not uncommon for a good roller canary to sell for $50. The comparatively new American industry of canary breeding has developed since the World War when European bird importations halted.

In overlooking the treatment of canaries many practitioners neglect an important phase of veterinary practice.

External Parasites

1. *Docophorous communis* or bird louse. This parasite feeds on the feather structures, irritates the skin, and causes discomfort to the bird.

Treat pediculosis with pyrethrum, using a small bellows. Repeat three times at intervals of a week to insure destruction of any young lice hatching in the meantime.

2. *Dermanyssus avium* or bird mite. Its natural color is white, but is nearly always filled with the blood of the parasitized bird giving it a bright red color. Habits are nocturnal. They are often found in cracks and crevices of the cage.

Control is achieved with use of coal oil, kerosene, or one ounce of crude carbolic acid in a gallon of water applied to the cage, or the mites and their eggs may be destroyed by immersing the cage in boiling water.

Ailments in General

Most diseases from which canaries suffer may be traced to some untoward circumstance in their handling. When these pets become sick the first step is to see that they receive a proper diet and that they are surrounded by general sanitary conditions. Where a number of birds are kept and a contagious or infectious disease breaks out, isolate the affected individuals. Move a sick bird to a warm place that is protected from drafts. Medication is best accomplished by administration of the medicine in the drinking water or with a medicine dropper.

Bone Fractures

Fractured bones are common in canaries. The prognosis is favorable if the break is recent, the soft tissues not lacerated and the fracture simple.

1. *Fractures of leg or foot.*—The bird is laid on the back and feathers clipped in the affected region. Cleanse and wash the skin and disinfect with Liquor Cresolis Compositus 3%. Reduce the fracture and wrap the part with a strip of adhesive tape, taking care not to draw the tape too taut.

Keep bandages in place for at least two weeks. Keep the bird isolated in a cage without perches and free of mites and lice. A tight bandage is quickly followed by a dry gangrenous process due to circulatory obstruction.

2. *Fractures of the wing bones.*—Treat as above except that the bandage should be longer and broader and pressed with the fingers into a flat shape corresponding to the shape of the wings. The bird is then wrapped in bandages so that both wings lie close to the body.

Cutaneous Emphysema

Emphysema of the skin refers to a condition manifested by an abnormal amount of air in the subcutaneous tissue. This condition is a result of a wound or spontaneous rupture of some of the air containing organs, e.g., trachea, lungs, air sacs, and their communicating structures.

Cutaneous emphysema occurs principally in young birds. Relief of the emphysema may be accomplished by incising the skin over the affected, swollen area, thereby preventing the accumulation of air.

Catarrhal Rhinitis

Catarrhal rhinitis is a mild inflammation of the nasal mucosa. No definite cause has been assigned to this condition. Weak, poorly nourished birds are most susceptible. Exposure to wind, rain, cold drafts favor its development.

Symptoms.—The symptoms of catarrhal rhinitis are: dullness, anorexia, dyspnea and unilateral or bilateral nasal discharge. The watery discharge may disappear in two to four days or may assume a more viscid consistency which may necessitate mouth breathing. The discharge may also escape through the mouth.

Morbid Anatomy.—The nasal mucosa becomes swollen, congested and an excessive secretion of mucus is poured out.

Treatment.—As the disease is of short duration, it is usually necessary only to place the patient under more favorable conditions to bring about recovery. In severe cases wash the nostrils daily with a 3% boric acid solution or a 1% creolin solution.

A useful preparation for internal use consists of oxymel of squill, glycerine, ipecacuanha wine, and sweet spirits of nitre, of each equal parts. Place 20 drops of this mixture in the drinking cup. A piece of bread scalded with milk, pressed nearly dry, sprinkled with sugar is an excellent food to give during colds and loss of condition. It should be given fresh every morning and the remnants removed in the evening.

Infectious Diseases

Infectious diseases of canary birds cause heavy losses in breeding establishments where hundreds of birds are kept in close contact with one another. These conditions and the introduction of fresh breeding stock furnish favorable circumstances for the spread of disease and severe losses occur.

Most infectious diseases of canary birds fall into one or the other of the following groups:

1. *Infectious necrosis* — caused by a member of the hemorrhage septicemia group.
2. *Bacillus paratyphus* B infection.

Infectious Necrosis of Canaries

Definition. — Infectious necrosis of canaries is a disease characterized by the presence of necrotic lesions in the spleen and liver. It is commonly called bird fever, canary fever, septic fever or bird plague.

Etiology. — *Bacillus canariensis necrophorus,* a non-motile, gram negative, bipolar-staining, rod 1.5 to 2.5 microns long, 0.5 to 1 micron in width. Some investigators classify it as a member of the hemorrhagic septicemia group.

Pathogenicity.—This organism induces the disease in canaries when introduced subcutaneously or per os. Per os, the incubation period is three days. Mice, sparrows, guinea pigs and rabbits are susceptible. Pigeons vary in susceptibility. Hens are resistant to this infection.

Symptoms.—No pathognomonic symptoms are seen. Anorexia, depression and squatting in a corner of the cage may be noted. Death occurs in 24 to 36 hours, after the first symptoms have been observed.

Morbid anatomy.—The most characteristic features at autopsy are the metastatic focal lesions in the spleen, liver, and occasionally in other organs. The liver is enlarged, contains numerous yellow nodules of the size of pinhead and larger as seen microscopically. A hand lens reveals smaller punctiform foci. The involved areas present a great variety of shapes and do not separate readily from surrounding tissue. They may be leathery in texture and crumbly in structure. The cut surface of the larger lesions consists of a homogeneous mass surrounded by an opaque, gray-colored zone. The spleen is swollen into a cylindrical shape and contains similar nodules. These distend the capsule and give the organ an irregular undulating appearance. The nodules are so numerous and the spleen so fragile in consequence that it breaks apart when grasped with tweezers. The pharyngeal mucosa occasionally contains yellowish nodules. The walls of the pleural cavities occasionally are covered with yellowish exudate and the lungs may be involved. Enteritis is sometimes observed. The necrotic lesions resemble those of avian tuberculosis but

the bacillus of tuberculosis is not demonstrable.

Microscopically the smaller nodules consist of great clumps of bacteria, whereas the larger lesions consist of clumps of bacteria surrounded by necrotic cells. The largest nodules consist of a number of smaller ones that have fused. The etiological factor is not constantly present in the heart blood.

Infection in Canaries Caused by Bacillus Paratyphosus B.

(Salmonella Schottmulleri)

Definition.—This is a specific infectious disease characterized by enlargement of the spleen and enteritis. It is commonly called septic fever of cage birds.

Etiology.—*Bacillus paratyphosis B* a motile, gram negative, amphitrichous rod demonstrable from fresh smears from heart blood or organs.

This organism is highly virulent for canary birds, mice, guinea pigs and rabbits.

Symptoms.—In the beginning of the disease the bird is not so lively as usual and sits on the perch with ruffled feathers. Later the head is turned backward between the drooping wings. Occasionally the bird hops about briskly and eats. As the disease progresses depression increases, appetite ceases entirely, droppings become thin, eyes are kept half closed, and respiration becomes quickened to 150 per minute. At times the bird peeps hoarsely. Movements become uncertain, the bird falls down, loses consciousness, and dies in a spasm.

Morbid anatomy.—Rigor mortis is pronounced. The legs are most often extended from the body. There is more or less marked enteritis. The spleen always shows hyperemic enlargement, and is two to five times its normal size. The liver and kidneys are hyperemic. Smears from the heart blood contain great numbers of short thick rods in pure culture.

Prophylaxis.—The first measure should consist in separating and isolating the healthy from the diseased birds. Remove and burn the droppings daily. Feed and water containers should be removed from the cages daily and should be disinfected.

Treatment.—Supply the sick birds with drinking water containing three to five drams of iron sulphate per liter. It is well to separate the affected birds so that there will be one bird to each cage.

A Septicemic Disease

(Observed by Freese)

Symptoms.—The bird at first ruffles the feathers occasionally and depression sets in. The next day it sits quietly on its perch, peeps now and then, and shows some dyspnea. Thirst increases but the appetite is not changed. There may be slight diarrhea. It is characteristic that the sick birds hop around in the cage at frequent intervals like healthy birds. The symptoms become more marked and death occurs in a day, or at most three days. Some hours before death the sick birds sit with ruffled feathers on the perch or in a corner of the cage. The eyes are half closed and the head is turned backward and held in the feathers. At this time the respiratory rate is greatly increased. The birds often continue to eat until within a few hours of death.

Morbid anatomy.—Blood is coagulated and dark red in color. The mucous membrane of the duodenum is swollen and diffusely reddened. The liver is either congested or fragile and yellow in color. In most cases the spleen is without microscopic lesions but occasionally there is hyperemic swelling.

Etiology.—Small rods, 0.5 to 1.5 microns in length and stain uniformly are seen in heart blood smears stained with the common analine dyes. These organisms are Gram positive, non-motile, facultative anaerobes. They are sparingly found in liver, spleen, and kidneys. Hens, pigeons, rabbits and guinea pigs are immune to subcutaneous injections of the heart blood of a canary bird dead of the disease. However, sparrows and mice are susceptible.

Septic Enteritis

Symptoms.—There is dullness alternating with periods of apparent complete health. There are frequent attacks of weakness, somnolence, with impairment of appetite and increased thirst. During the last few days the bird sits in the bottom of

the cage with eyes closed. Death occurs in 10 to 12 days.

Morbid anatomy.—When the disease is of long duration the carcass shows great emaciation. The breast muscles are yellow as if cooked. The spleen and liver are always enlarged. The kidneys and heart muscle are yellow and clouded. Rarely, there is a marked intestinal hyperemia. The respiratory organs are normal; the brain anemic.

Etiology.—The *Bacillus loxiacida* is constantly found in the spleen, liver and blood of canaries suffering from septic enteritis. It is a motile, gram-negative rod.

BIBLIOGRAPHY

1. Gray: Disease in canaries. *Vet. Rec.* Vol. 22, 1910, p. 753.
2. Gilruth: Diseases of canaries. *Vet. J.* n. s. Vol. 17, 1910, p. 655.
3. Cornell: Infectious Necrosis of canary birds. *Vet. J.*, 84:350-6, 1928.
4. Ward and Gallagher: Diseases of Domesticated Birds. Macmillan Co., N. Y., 1923.
5. Wetmore: Canaries, their care and management. U. S. D. A., 1923.
6. Stroud: Diseases of Canaries. Canary Publishers Co., Kansas City, Mo.
7. Claude St. John: Bird Ailments and Accidents. Cage Birds Publishers, London, Eng.

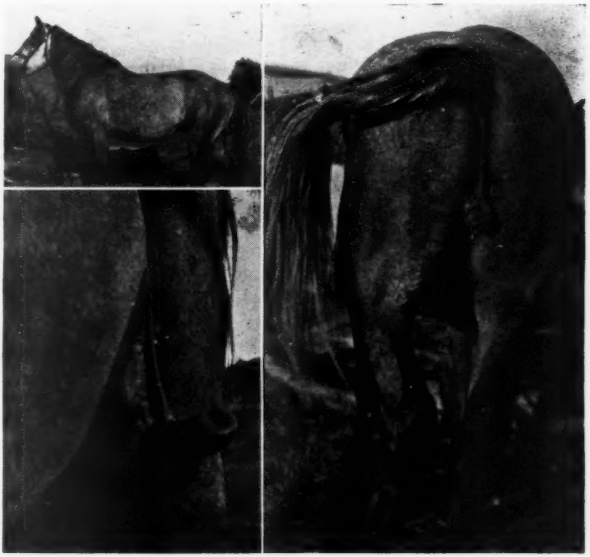
CANARY BIRD PERIODICALS

1. *Canary Journal,* Kansas City, Mo.
2. *American Canary Magazine,* Chicago, Ill.
3. *Cage Bird World,* Baltimore, Md.

Personnel of the Federal Bureau of Animal Industry

At the end of the year (June 30, 1937) the Bureau rolls showed 4,012 regular employees of all grades and designations in Washington, D. C., and in the field, as compared with 4,161 at the beginning of the year. There was thus a net reduction of 149 in the total number of regular employees. Additions consisted of 424 new appointments, 19 transfers from other bureaus or departments, and four reinstatements, a total of 447. Separations consisted of 77 resignations, 33 deaths, 16 transfers to other bureaus or departments, two removals for cause and 66 retirements under the provisions of the amended Retirement Act of May 29, 1930. Other separations, principally of agents, unskilled laborers and seasonal and temporary employees, numbered 402, a total of 596.

In addition to regular employees, there were at the beginning of the year, 2,550 emergency employees of various grades and designations serving in various activities, principally campaigns against bovine tuberculosis and Bang's disease. During the year there was an increase of 295 in the number of emergency employees.—*Rpt., Chief, B. A. I., U. S. Dept. of Agric.*, 1937.



A four-year-old hermaphrodite horse presenting an almost exact neutralization of male and female sex organs. This animal is inclined to be a bit excited among mares but is ordinarily as gentle as any farm horse. The mammary development is that of an ordinary 4-year-old mare. There are no testicles apparent, but I did not make a rectal examination. Externally there is a vulvar fold, through which extends a rudimentary penis. The urethral opening is normal, except it is dorsal due to the position of the penis. When this colt was foaled the owner called me to determine if possible what sort of creature he had. The dam has had two foals since. Both were males with double inguinal hernias. Covered operations were done on both, and they have developed well. The illustration in the lower left shows the penis erected, due to proximity to a mare in estrum. —*L. L. Howell, Belmont, N. Y.*

Acute Carbon Monoxide Poisoning in a Dog

By JOSEPH R. STERLING,
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Kansas State College '38

IN WRITING an article on a particular disease, it is customary to begin with a resume of previous work on that disease. In this instance, we have been unable to find any detailed report on carbon monoxide poisoning in domestic animals. Nor do the textbooks furnish any information.

Carbon monoxide poisoning is a result of inhaling the toxic gas with inspired air. Carbon monoxide has an affinity for hemoglobin 300 times as great as oxygen. It combines with the hemoglobin of the blood to form carbon monoxide-hemoglobin, which destroys the efficiency of the blood as an oxygen-carrying medium. Thus, the tissues are deprived of oxygen in direct proportion to the amount of carbon monoxide inhaled. It is quite generally believed that all the damage that results from gas poisoning is produced while the victim is breathing the gas. Most authorities state that little or no damage occurs after the patient has inhaled a few breaths of fresh air.

There are two theories as to the mode of action of CO on the nervous system, viz: (1) that the anoxemia due to the combination of carbon monoxide and hemoglobin results in deprivation of oxygen to the nervous tissues with consequent nervous changes; (2) that the carbon monoxide, after having saturated the erythrocytes, becomes dissolved in the blood serum and attaches itself to the nerve cells to produce a direct toxic effect on them. However that may be, it is a fact that very low concentrations of CO are highly dangerous. Thus, a concentration of as little as 0.5% carbon monoxide in the atmosphere of a limited structure will prove rapidly fatal to animal life.

The following case was observed, throughout its course, by the writer, during the summer of 1937, at the American Hospital and Home for the Care of Pet Animals in New York City. The patient was under the care of Drs. Purmell and Klausman and the writer. The affected dog was a male, liver-and-yellow, springer spaniel, two years and three months of age and weighing 38 pounds.

August 2, Mrs. S., owner of the patient, left her apartment to do some shopping. As was her custom, she left the dog in the kitchen. The kitchen window was open and the door was closed. Returning at approximately 5:00 p. m., Mrs. S. was almost asphyxiated when she opened the kitchen door due to the high concentration of CO in the atmosphere. Quickly realizing the dog's plight, she managed to pull him out of the apartment and carry him up to the roof.

It was noted, at the time, that there was much vomitus, feces and urine on the kitchen floor—indicating that there had been a general relaxation of the sphincter muscles during the process of gas poisoning. Gas was found to be coming from an open, unlighted jet in the kitchen range. It must be concluded from all the circumstances that the dog himself must have opened the gas jet, possibly in searching for food.

The high toxicity of the illuminating gas used in this apartment house lends credence to the supposition that the jet must have been turned on only a short time before Mrs. S. returned to her apartment. According to officials of the company which supplies the gas, is is a mixture of carburetted water gas and coke oven gas with a CO content always about 25%. Thus, we can see that the dog would have been killed almost instantly if the kitchen had been air-tight, or even fairly air-tight. Fortunately, the kitchen window had been left open. Still, the instantaneous effect of the gas on the dogs' rescuer leads to the conclusion that the CO concentration was fairly high, and that the patient must have breathed the gas for only a very short time.

After about five minutes on the roof Mrs. S. realized that the dog's condition was not improving and decided to take him to a veterinarian. The patient entered the Ameri-

can Hospital on August second, at 5:25 p. m. At that time, examination disclosed the following: temperature, 99° F.; pulse, barely perceptible, and respiration apparently arrested. The patient was completely unconscious, the eye reflexes in abeyance, and the mucous membranes were cold and white. Thus the condition had reached the pallid or second stage which sometimes follows the cyanotic or first stage.

Artificial respiration was resorted to immediately, together with the administration of aromatic spirits of ammonia by inhalation and the injection of 0.75cc of epinephrin subcutaneously. These measures served to revive the patient somewhat and he was then given a pint of warm, black coffee per rectum. This was followed by an intramuscular injection of 5cc of camphorated oil to produce prolonged stimulation.

The patient was then transferred to the roof of the hospital to provide plenty of fresh air and massage was applied to aid circulation. He remained on the roof for several hours, being kept warm by the use of blankets and hot water bottles.

During the period on the roof the dog showed distinct signs of improvement and he was hospitalized. During the night, a close watch was kept on the patient and, since he seemed to be steadily improving, no treatment was given except rest, fresh air, and warmth. On the morning of August 3rd, 15 hours after the patient had entered the hospital, he was no longer in a state of stupor but seemed definitely on the way to recovery. From then on, treatment consisted wholly of rest and fresh air, and concentrated, nourishing food, given in small quantities at frequent intervals.

By August 5th the patient seemed almost completely normal and he was discharged at 6 p. m. At that time, his condition was as follows: temperature, 101° F.; pulse beats, 75; respiration, 18; breathing, slightly laborious; eye reflexes, normal; sight, normal; mucous membranes, normal. Further he was lively and showed a constant desire to run and play.

August 8, at 11 a. m., the patient was returned to the hospital for further treatment due to the fact that he had suffered a severe relapse. At that time, he exhibited the following: temperature, 100.2° F.; pulse, very weak, 52. respiration, 34; severe dyspnea. He was very dull and showed hypoesthesia and was practically blind. If petted, he would wag his tail a few times but would retain his lateral recumbency. He was unable to stand and would not move unless forced to do so. We could

"Bozo's Marol Binker," the victim in the carbon monoxide poisoning described in this report.

not obtain an eye reflex by use of a moving object unless the object was within two inches of his eyes. In one test, we set a bowl of water one foot in front of him at a time when he was obviously thirsty but he seemed to be completely unaware of the presence of the water. Examination of the eyes failed to disclose any lesions. He drank water voraciously when his mouth was put in contact with it and he defecated and urinated regularly. He ate with little show of appetite. However, he did consume almost as much food as a healthy dog and at no time did he show any tendency towards emaciation or dehydration.

From August 8th to 10th, the patient was allowed as much fresh air as possible together with rest, quiet and nourishing foods. During this time, no improvement in his condition could be noted. On the night of August 10th, at the request of Mrs. S, the dog was discharged from the hospital.

From the tenth to the twelfth of August,

the patient was kept in the client's house and its condition seemed to be static. Then, on the thirteenth, a very significant change in the course of the case occurred. On the morning of that date, Mrs. S. awoke to find her dog as lively as he had ever been. She brought him, once more, to the American Hospital where an examination showed him to be in an apparently normal state of health, except for a case of eczema due to flea infestation.

At that time, he showed the following: temperature, 101.2° F.; pulse, 86; respirations, 24; no dyspnea; eyesight, completely recovered; mucous membranes, normal; locomotion, normal. According to the owner the dog's state of health seemed to be exactly as it had been previous to the unfortunate accident. In her own words, "the dog is as fine, lively and intelligent as ever." Since that time, no further relapses have occurred and it seems reasonable to conclude that he has completely and permanently recovered.

The above is but one case of carbon monoxide poisoning. Therefore, though the patient recovered, we do not know the real value of the symptomatic treatment outlined herein. However, it seems a logical method of treating carbon monoxide poisoning and well worthy of trial.

As has been previously mentioned, veterinary literature on the subject is lacking. Therefore, we must take recourse to books on human medicine for further possible aid. A few extracted pointers, particularly on treatment, seem worthy of mention. According to Sajous (Analytic Cyclopedia of Practical Medicine), direct transfusion of whole blood is of great value. Also, intravenous injections of hypertonic saline solutions have given good results in attempts to reduce the high intracranial pressure that occurs in carbon monoxide poisoning. In quite a few cases, venesection accompanied by injection of normal salt solution has been employed, and has been followed by rapid recovery. Methylene blue solution (1% intravenously) has been used, but its value has not been determined.

J. C. DaCosta (Modern Surgery), together with other authorities on the subject, recommend the administration of oxygen-carbon dioxide mixtures as the most valuable single treatment of carbon monoxide poisoning. According to DaCosta, the most efficient method for the elimination of carbon monoxide from the blood stream consists of the inhalation of a mixture of 90 to 92% oxygen and 8 to 10% carbon dioxide. This mixture produces five to six times as rapid elimination of carbon monoxide as does normal air. The average veterinary practitioner rarely sees a case of accidental carbon monoxide poisoning, although it is a common means of euthanasia. Therefore, the purchase of an inhalator apparatus is economically unjustified. However, it is well to keep the above treatment in mind as human inhalator facilities are sometimes available to veterinarians, and the use of such apparatus would be well justified in some of our valuable domestic animals. Furthermore, one authority definitely states that the human oxygen masks can be used readily on domestic animals.

—Photo by H. Selvidge.

Albino Buck at the State Game and Fur Farm, Wisconsin Department of Conservation, Poynette, Wis.

Longevity of the Whipworm

Very little seems to be known about the number of weeks that any of the helminths normally live in the dog. It is a subject which should be most carefully studied, because of the value which such a study may be to the owners of dogs and their advisers, the veterinarians. While the subject of this paper deals with the life duration of the whipworm, there is no thought on the author's part that he has determined the exact length of life of any individual worm, but rather that his observations may at least be of help to others who may, by more exact methods, be able to carry the study further.

Periodic fecal examination has been a routine procedure in the author's experimental kennel for many years. Several times during the summer, the feces of every dog, with the exception of puppies, are examined for parasite eggs. The feces of the puppies are examined as a composite specimen for each pen. In general, the last examination is made in the late fall, after the ground has frozen. The first examination is made in the early spring. Any exceptional dogs are examined before and after treating, when it has been found that treatments are in order. Some dogs are thought to have vomited anthelmintics and these are also re-examined.

During the summer of 1934, a good many new dogs were added to the kennels. These showed whipworm infestation in a large percentage of cases. A few were severe, but most of them showed only minor infestations. The severe cases were treated with santonin and calomel, which reduced the severity very markedly. The rest were not treated unless they had hookworms also. These were given tetrachlorethylene, which had but slight effect in reducing the numbers of whipworms so far as one could determine from the numbers of their ova in the feces.

In the spring of 1935, fecal examination revealed no evidence of whipworm. This was puzzling; therefore, during the late fall of 1935, a most careful check was made and it was found that there were eleven dogs in the kennels suffering from varied infestations of whipworm, ranging from mild to severe. Nothing was done to remove them, so far as the administration of anthelmintics was concerned; but the feces were removed daily from the kennel runs, except where snow made it impossible. Bedding was changed as soon as it became muddy or damp, it was virtually impossible for a dog to bring whipworm ova or ova from any parasite into the house on his feet or in his hair, and lie on them long enough for body heat to incubate them, to the infectious stage. In the cold these ova cannot incubate to become infectious.

In the spring of 1936, therefore, it was most interesting to note that not one of the eleven dogs which had showed whipworm eggs in the feces in the late fall, showed a single whipworm ovum. The study was conducted with the utmost care.

In the late fall of 1936 another check was made. Six dogs showed infestation. As I write this, on February 15, 1937, I can report that a fecal examination made today shows that only two of the dogs have mild whipworm infestations and the other four are negative. This seems quite remarkable when it is realized that in Orange, Connecticut, where the kennel is located, we have had very little snow all winter. The ground has been muddy a good deal of the time. The bedding has had to be changed sometimes several times a week, because of the mud tracked into the houses.

That some condition other than the age of the worm has caused this natural eradication, is doubtful. The food fed during the summer and winter has been the same. Tetrachlorethylene given in the summer did not eliminate the whipworms, but during the winter, these helminths disappeared, with no vermicide treatment. The logical explanation would seem to be that the worms harbored by these dogs last fall, have since died of old age, and no new infestation has occurred because of the cold weather, which did not permit incubation of ova. The frequent change of bedding prevented the incubation of ova by body heat. It would seem, therefore, that the whipworm has a rather short life (possibly a long one considering an animal of its size). The writer does not know the

length of its life in the sexual stage, but he feels that the above experience justifies a tentative conclusion that it does not exceed five months and possibly may be shorter.

LEON F. WHITNEY.

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* * * *

Milk Fever Today and Yesterday

Thirty to 40 years ago the fatalities from milk fever were many, even with the best treatment then known. The mortality was particularly high if the attack occurred when the new calf was only a few hours old. If the attack occurred after the calf was a day old, the prognosis was more favorable.

When the treatment by inflation of the udder was adopted, the results were remarkable. At this time oxygen was used to inflate the udder, and infection of the udder was rare or unknown. Later air instead of oxygen came into use, and with it came the layman, first with his bicycle and later with his automobile tire pump, and mastitis following the treatment became common. There were always a certain percentage of cases that required repeated inflation. When oxygen was used, this occasioned no harm, but with air and the tire pump, such cases were prone to infection, and mastitis might almost be said to have been a routine sequel in prolonged cases.

With the advent of the calcium treatment, recoveries reached practically 100%. Calcium chloride is the preparation to give if it is administered properly. In a large number of cases I have had only three abscesses as a result of its use, and not a death from shock. I always administer 25cc of camphorated oil intramuscularly previous to any intravenous injection. Of course this procedure is followed in the intravenous injection of calcium chloride and probably, in part, accounts for the fact that I have had no deaths from shock in the large number of cases in which I have used it.

The effect of calcium chloride is more prompt than the effect of calcium gluconate, and there are fewer relapses. However, relapses will occur and require a second treatment, and even additional treatments, no matter what agent is used, oxygen, air, calcium chloride or calcium gluconate. It is quite common to be called to these cases 12 to 18 hours after they have gone down and after the owner has given a drench, which is occasionally fatal. The cases in which treatment is delayed are more prone to recidivation than are cases that are treated promptly. A second treatment, whether it be inflation or intravenous injection should not be given in a shorter period of time, six hours after the first.

There are many cases of what might be spoken of as marginal milk fever that the owner does not recognize. These animals do not go down, but just become slightly wobbly and staggering, and they may continue this way anywhere from two or three days to as many weeks, and while such animals usually, ultimately recover, the dairyman's loss in milk production is considerable. Such cases respond splendidly to calcium gluconate treatment, either intravenously or intramuscularly. Recovery is usually complete in 30 minutes, and relapses are few.

During the past 18 months I have had more cows go down a second time 12 to 24 hours after treatment (which at the time was apparently successful) than in all my previous experience. A few of these cases have gone down on three successive days, and one had the fourth attack on the fourth day. Each time after the injection of 250cc of calcium gluconate, intravenously, these cases were up in 20 minutes, and all eventually recovered. After a good lot of experience of this kind, I began giving a copious enema, and two pounds of epsom salts through the stomach tube as soon as the cow was well on her feet. I have not had any more of these relapses after 12 to 24 hours where this follow-up treatment was used.

I tried the intravenous injection of glucose solution as has been advised by some to prevent these relapses; tried it in three cases. There were no relapses; the animals all died immediately.

Because of the greater care necessary in giving calcium chloride and the longer time required for the intravenous injection, of

late I have been using calcium gluconate, and my recoveries have been more than 95%. When I strike a very stubborn case that has received two injections of calcium gluconate and is not up in 12 hours, I resort to calcium chloride, which I believe to be the surer remedy. However, there is an occasional cow in which recovery starts in say 30 minutes after the first injection and progresses to where the animal rolls up on her sternum, looks bright and eats and drinks normally, but will not stand. If repeated intravenous injections do not get such animals on their feet in 24 hours, I get out the oxygen tank, and the results are usually all that could be asked for. I do not use—I have never used—the tire pump and air for inflating a cow's udder, and while I do not inflate udders now in 1% of my cases, if I do use inflation, I believe in doing it right, and it is done right when the udder is inflated with surgical cleanliness and oxygen used.

L. E. THOMPSON.

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Indigestion in the Dairy Cow

Stasis of the rumen in dairy cows, called by some impaction, by others overloaded paunch, has been called to my attention frequently in the past 22 years, during which I have practiced in one of the largest of dairy counties. It is usually the result of dietary errors.

The symptoms include suspended rumination, greatly reduced milk flow, little if any bowel movement, refusal of all feed and an inclination to stand with hind feet in the gutter. The rumen is usually full and bulges out the left flank, has a doughy feeling on palpation and tympany may be present.

Treatment.—The treatments are many. Here is one I have used and find very satisfactory:

℞ Strychnine sulphate ..... 2 gr.
Capsicum ..... 2 dr.
Ginger ..... 2 dr.
Ammonium carbonate q.s. a.d. to fill a one and one-half-ounce capsule.

Give at once and with it two one-ounce capsules of oil of turpentine, and massage the rumen for about five to ten minutes.

Leave the following with the owner to administer:

℞ Powdered nux vomica
Powdered gentian
Powdered ginger aa..... ℥iss
M. Sig. One tablespoonful on tongue or in feed every three hours.

This treatment not only brings the cow back to normal flow of milk, but it makes a hit with the owner. They pass some very fine comments on it. It beats drenching; it beats passing the stomach tube, and when the year is up, you have had more calls for so-called impaction of the rumen, and many more recoveries.

All one has to do is read, in Hoare's book on veterinary therapeutics, the discussion on epsom salt, oils, and ammonium carbonate and strychnine sulphate; then ask does one still want to continue using epsom salt. The answer is, no.

In giving the capsule, one wants an 18-inch balling gun, so one won't skin one's fingers against the incisor teeth. It also does away with the farmer drenching his cows with epsom salt. It is a practice builder.

Small cows, say yearlings or Jerseys, can take a Reeks' colic capsule very well.

Of course, after treatment and care of the animal play an important part in bringing about a recovery.

A colleague told me some time ago that since he has used this treatment that I recommended to him, he has not lost any cows.

This treatment also works fine in bloat and in engorged rumen from eating too much food; a thing which happens in the fall of the year, from getting into green fields, or eating too much grain.

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A Study of Experimental Streptococ

STREPTOCOCCIC mastitis is a major dairy problem from both the economic and hygienic viewpoints. Shaw and Beam[5] reported that quarters infected with streptococci produce approximately 22% less milk and 24% less butterfat than the streptococcus-free quarters. Since many cows are infected in all four quarters a similar decrease in milk and butterfat production may be expected and has been observed among dairy cows. Minnett and Martin[4] studied the milk yield of mastitis-infected cows and reported that the reduction of milk yield due to mastitis was 954 pounds per lactation period. More recently, White, *et al.*,[6] reported the reduction of milk yield of mastitis-infected cows as being 425 to 563 pounds per lactation period. It is obvious that the milk production of mastitis-infected cows is reduced as compared to the production of mastitis-free cows, even though investigators do not agree on the exact extent of the reduction.

Bryan and Trout[3] found that milk produced by cows with steptococcic mastitis was inferior in quality as determined by flavor, chloride content, leucocyte content, bacteria count and methylene blue reduction time. Ninety per cent of the milk samples from infected cows in one herd were criticized as having a salty flavor, while only 14% of the milk samples from a herd free of streptococcic mastitis were so criticized. These latter animals were near the end of their lactation period when such changes may normally occur. Only 50% of the steptococcus-infected cows produced a class one (good) milk as judged by the methylene blue reduction test, as compared with 98.5% of the non-infected cows. Similarly, 68.5% of the infected cows produced milk with a standard plate count of more than 1000 as compared with 5.8% of the non-infected cows. The decreased production and lowered quality of milk produced by cows with streptococcic mastitis are sufficient reasons for emphasizing the economic side of the problem. In addition, the hygienic aspects of the problem are important.

The streptococci that cause mastitis have been found to be a cause of human infection. These include the steptococci of human and bovine origin that cause mastitis. The human infection usually takes the form of septic sore throat epidemics when human strains are concerned and isolated cases of sore throat together with further complications when steptococci of bovine origin are concerned. Brooks and Tiedeman[1] present evidence that milk from mastitis infected cows may cause human "distress," especially in babies, as a result of the toxins present in the milk. The economic and hygienic aspects of the problem are greatly emphasized when the high incidence of this cattle disease is considered.

Eighty-six per cent of the herds and 26.2% of all milking cows were found to be infected with streptococcic mastitis in a survey conducted in a typical milk shed.[2] Figures dealing with the incidence of streptococcic mastitis, as reported by other investigators, are presented in the above paper. These data, together with the human health problems involved, make this one of the, if not the, most important dairy cattle disease at the present time.

The importance and wide spread of streptococcic mastitis create a big problem with respect to the control or eradication of the infection. It is essential that such recommendations be based on an understanding of the following:

1. The manner of spread of the streptococcus from cow to cow or from some other source to the cow.

2. The portal of entry of the strepcoccus into the udder.

3. The time when most spread of infection occurs. A great deal of this information can be gained only from a study of experimentally induced streptococcic mastitis.

A study was undertaken to determine:

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[1] Journal Article No. 305, new series, from the Michigan Agricultural Experiment Station.

[2] Excerpt from a thesis submitted to the Graduate School of Michigan State College in partial fulfillment of the requirements for the degree of doctor of philosophy.

cic Mastitis in Dairy Cattle

(1) The portal of entry of the streptococcus into the udder; (2) the possibility of controlling streptococcic mastitis by segregation of the infected cows and the employment of sanitary procedures in handling the herd; (3) the variation in composition of the milk following infection by the streptococcus; (4) the rapidity of the reduction in the quality of the milk after infection; (5) the value of the various tests in the detection of experimentally induced streptococcic mastitis (a) direct-microscopic test, (b) indirect tests, and (c) physical examination of the udder; (6) the normal cellular and hemoglobin content of the blood and variations following exposures and infection, and (7) the histopathology of the udders when the duration of infection is definitely known.

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Summary

A typical strain of *Strep. agalactiae*, recently isolated from a case of chronic mastitis, was used in experimentally inducing streptococcic mastitis in normal cows, i.e., cows that were free from tuberculosis, Bang's disease and streptococcic mastitis.

The cows subjected to experimental exposure became infected only after repeated exposure by dipping the uninjured teats into the culture or after dipping of injured teats into the culture. When the teats were injured, sufficiently to draw blood, infection developed as a result of one exposure. Repeated exposure of the cows to the streptococcus per os, or by the subcutaneous or intravenous routes did not result in streptococcic mastitis. Of the six cows used in these experiments four became infected by dipping the teats into the culture, one by dipping the lacerated teat into the culture and one (the control cow of the second year) with no experimental exposure but exposed to the streptococcus as a result of being stabled in the same barn with the infected cows.

These results indicate the difficulty of controlling streptococcic mastitis within a herd, even though the infected cows are segregated apart from the non-infected cows and sanitary procedures are employed in handling the dairy herd. Such procedures are of value in the control of infection since the control of the first year's study was protected from infection by these means.

The casein nitrogen, whey nitrogen and

Courtesy, B. A. I.

Crossbreds for Market Lamb Production
From Hampshire rams and Rambouillet and Corriedale Ewes. U. S. Sheep Exp. Station, Dubois, Idaho

curd tension, leucocyte content, pH and chloride content of the milk did not vary greatly prior to infection. In general, following infection the casein nitrogen and curd tension of the milk were decreased, while the other determinations gave increased values with respect to the composition of the milk.

The quality of the milk produced, following infection, was greatly reduced as measured by the standard plate count, and the methylene blue reduction test. Three of the cows gave milk with a bacteria count of greater than 1000 per cubic centimeter during the first month after infection, two in the second month and one in the third month. The methylene blue test rating of the milk dropped from class one to class two, three or four within one month after infection in four cows, within two months in one cow and within three months in one cow.

The value of the various tests in detecting experimental streptococcic mastitis, in decreasing order of efficiency, are: 1, Microscopic test; 2, physical examination of the udder; 3, leucocyte count of milk; 4, chloride content of milk; 5, thybromol test (pH), and 6, physical examination of the milk.

Exposures of cows to *Strep. agalactiae* and infection of the udder by this organism did not alter the cellular or hemoglobin content of the blood. The normal values, for the cows studied, are presented.

Exudative processes were present in all twelve quarters of the three experimental cows slaughtered, with productive processes in 10 and fibrosis in five of the quarters. In all quarters the processes of plane three were of longer duration than those of plane two and of plane two than those of plane one. The evidence presented indicate that streptococcic mastitis may be considered as a chronic but progressive condition.

References

1. Brooks, Paul B., and Walter Von D. Tiedeman, 1937. Relation of Bovine Mastitis to Human Disease; *Amer. Jour. Pub. Health,* 27:4, pp. 338-41.

2. Bryan, C. S., 1937. The Incidence of Streptococcic Mastitis Among Dairy Cattle; *Vet. Med.,* 32:2, pp. 70-74.

3. Bryan, C. S., and G. M. Trout, 1935. The Influence of Streptococcic Infection of the Udder on the Flavor, Chloride Content and Bacteriological Quality of the Milk Produced; *Jour. of Dairy Sci.,* 18:12, pp. 777-92.

4. Minett, F. C., and W. J. Martin, 1936. Influence of Mastitis and of *Brucella abortus* Infection upon the Milk Yield of Cows; *Jour. of Dairy Res.,* 7:2, pp. 122-45.

5. Shaw, A. O., and A. L. Beam, 1935. The Effect of Mastitis Upon Milk Production; *Jour. of Dairy Sci.,* 18:6, pp. 353-57.

6. White, G. C., et al., 1937. Chronic Bovine Mastitis and Milk Yield; *Jour. of Dairy Sci.,* 20:4, pp. 171-81.

Courtesy, B. A. I.

Columbia Ewes, United States Sheep Experiment Station, Dubois, Idaho

Avitaminosis-A and Distemper in the Dog*

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The dog and the dog family, *Canidae,* before domestication were nocturnal and seminocturnal animals. Like other night-prowling carnivora, and beasts of the jungle, they attack and eat their quarry in darkness, resting and sleeping during daylight hours. That these animals should readily get about in darkness, nature has endowed them with a keen sense of night vision.

The faculty of night vision, common to all night-prowling beasts, is dependent to a great extent upon a liberal supply of vitamin A in the diet. It is this vitamin that is so vital to the retinal rod cells if this night vision is to be maintained. As is known, the vitamin A is synthesized by the rod cells in the light-perceiving layer of the retina into visual purple, the night seeing agent. When the eye is at rest in darkness these cells in the fringe of the retina continually regenerate and store this synthesized vitamin A (visual purple) and when the eye is exposed to light it undergoes a photochemical reaction, or bleaching process, in which carotene is split off. This carotene is a peculiar yellow pigment with the chemical formula: $C_{40}H_{56}$, and is commonly known as "pro-vitamin A". Just what part this fluorescent pigment plays in the faculty of night vision in the animals' eyes is not fully understood, but it is known to play some part in the eye's adaption to faint illumination.

All night-prowling animals are flesh-eating animals. It is from this source they obtain the much needed vitamin A (in freshly killed meat, the entrails, and glands.) If for some reason there should be a deficiency as a result of starvation or some body defect such as disease, intestinal parasites, etc., proper synthesis and assimilation of this vitamin could not take place and signs of body dysfunctions would appear as the result of the deranged functioning of the whole organism.

*Excerpts from an article in the Medical Record, Sept. 16, 1936.

Symptoms of Nutritional Disturbance

In the studies made on young dogs that had been deprived of vitamin A foods over a period of time, certain signs of these body dysfunctions were observed. One of those first noted was the inability of the animals to get about in darkness as readily as before. In fact this sign of night-blindness (hemeralopia) along with that of anorexia, were the prime nutritional defects detected when the dogs passed into the zone of sub-optimal nutrition. In the latter stages it was not difficult to detect other symptoms of the nutritional disturbance, the commoner ones being:

More or less congestion of the conjunctiva, the eyes becoming glassy and watery, later the discharge turning purulent, which matted the eyelids together.

The natural gloss was lost early to their quickly-shedding coat, and a papular rash made its appearance on the body, being more prominent on the ears, eyelids, and abdomen.

The skin desquamated in flaky particles, throwing off an obnoxious musty odor. This same odor was noted also in their secretions.

The cervical glands became markedly enlarged, and at times went on to pus formation.

Coryza, sneezing and trembling were more or less in evidence, as was gastrointestinal distress and pneumonic congestion.

Severe weakness and partial paralysis developed in their hind quarters, making standing and walking difficult at times.

Gudjonsson reported similar findings (though his studies were on the rat). He gave an excellent description of the common sequence of the symptoms of extreme A-avitaminosis which appeared to be the same as those noted in the dog suffering from the same disturbance.

When one compares the symptoms of A-avitaminosis to those of distemper in the dog, a striking similarity will be noted.

Were it not for the fact that distemper has a more rapid onset, one would have considerable difficulty in telling them apart, and conclude the two to be the one and same disorder. Later and more intensive research may show distemper commences over a much longer period of time than generally supposed, and that its etiological factor is a nutritional disturbance in which the antiinfective vitamin A, through some destructive process or faulty synthesis, falls considerably below the optimal zone and the dog becomes a victim to the early inroads of the distemper virus and other infective organisms.

Addendum

When the ill-fed dogs were placed back on a mixed diet with such added foods as liver, kidney, egg-yolk, cod liver oil and the other vitamin A containing foods, they would quickly sense out, and ate with relish, these special foods, and ignored entirely those that were lacking in that particular vitamin. What this special "sense" or instinct is that is common to animals that cause them to seek out those special foods so vital in maintaining the proper nutritional rhythm is not known, but it certainly is lacking in man, who will eat and drink anything—so long as it appeases his hunger, and slakes his thirst.

Some interesting data are being obtained in preliminary studies made on the cat. This animal, and the family *Felidae,* are conspicuously carnivorous in their diet. The household cat, though thoroughly domesticated, retains many characteristics of wildness, especially in its private hunting expeditions, nocturnal amatory wanderings, etc. When turned out into the woods it readily adapts itself to this wild life. In cats, the sight (night vision), hearing, and touch are even more highly developed than in the dog. For these purposes they evidently require a greater abundance of the vital food constituents (vitamin A, etc.). In addition to their carnivorous diet, they also eat fish whenever they can get it. The explanation of the better night vision in the cat is that there is greater dilatability of the pupil which enables it to make the most of feeble light. Also, there is a marked difference in the fundus oculi of the dog and cat. Whereas in the former the fundus is divided into two distinct parts, there is no such division in that of the cat. The tapetum nigrum is lacking, the whole fundus appearing as one complete tapetum lucidum, the colors even being more brilliant, and with a greater fluorescence than that seen in the dog. Apparently a greater amount of the night-seeing agent, the visual purple, is to be found in the cat's eye, thus accounting for their excellent night vision.

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Breeding Problems With Angora Goats*

The goat performs two useful functions. It cleans up brush, thereby saving its owner a great deal of work; and through its digestive process, it converts the brush into food and clothing for man, in the form of milk, meat, mohair, and skins.

The domestic goat is probably descended from the pasang or Greek ibex of the Near East. The long-haired Angora was originally developed by the Turks centuries ago. The first importations to Europe were apparently made in the sixteenth century, and to the United States as late as 1849. South Africa, Turkey, and the United States are the leading producers today. There are now over three million Angoras in this country, and the industry is concentrated chiefly in the Southwest, with Texas well in the lead. Of the 15 million pounds of mohair produced in the six leading states in 1935, Texas produced 13 million.

Improvement of the Angora goat has been entirely in the hands of private breeders. In the range herds, it has been carried on by the method of breeding high-grade does to registered bucks that conform to the standards of the breed association. Very little research work on breeding or genetics has been done by public institutions. The Texas Agricultural Experiment Station has been the leader in this field, and at its branch station near Sonora it is now carrying on three active projects on inheritance of type, inheritance of cryptorchidism or

*Summary of an article in the 1937 Year Book of the United States Department of Agriculture.

undescended testicles, and cytological and hybridization studies.

The best present representatives of the breed produce up to 75% more mohair than the average for the country. But there is a very great lack of uniformity even in good herds. If further progress is to be made, a research program will be needed to work out better methods than those now in use to determine an animal's inheritance, and a far better knowledge of the mode of inheritance of characters concerned in the production of good fleeces. Research usually pays well, but there are many difficulties and complications involved in such a program, and it can be carried on only with the active encouragement of the industry.

Meanwhile certain practical steps might be taken by the industry that would tend in the right direction. Among these would be a system of pedigree recording based on production of mohair, with certification of the records, as with dairy cattle and poultry; the selling of mohair on a quality rather than a weight basis, to stimulate improvement of the breed from this standpoint; and the adoption of different methods of awarding prizes at shows, on the basis of get of sires and outstanding families rather than individual appearance—a practice that would be in line with the use of the progeny test, which is overwhelmingly important in evaluating breeding stock, whether plant or animal.—*W. V. Lambert.*

* * * *

Thoroughpin Operation

Indication.—Bursitis serosa calcanei complicated with sclerosis of the bursal wall and fistulous thoroughpin which does not heal.

Instruments. — Scalpel, probe-pointed knife, curette, fenestrated curette, rat-tooth forceps, hemostatic forceps, wound retractors, lock forceps, scissors, catgut, silk suture, Esmarch's tourniquet, gauze, bandage.

Technique.—The horse is cast on the side opposite the affected leg. The skin in the region of the swelling, and considerably beyond it, is shaved. It is expedient to shave the area before casting, if practicable. The skin incision is made on the lateral side of the swelling and carried out to form an arch. The center of the incision is deepened until the bursa is opened. The opening is now extended upward and downward. The bleeding vessels are ligated; all fibrous clots are removed from the bursa, and all fibrous granulations are trimmed out with scissors. The rear bursal wall is scraped thoroughly with the curette, but the anterior wall is scraped only lightly, care being taken that all tissue particles are removed. Hemorrhage into the bursa is slight, and does not call for special methods of control. When the posterior wound lips have been trimmed carefully, the wound is then closed by means of sutures, and covered with the following paste:

℞ Bismuthi oxyjodati subgallici
Glycerini
Mucilaginis acaciae ..... aa 10.0
Kaolini 20.0
M. ft. pasta.

Technique II.—When the horse is cast a tourniquet is placed around the leg. The field of operation is prepared as for the previous operation. The bursa, however, is not opened. The skin is incised and peeled back until one can grasp the bursal wall with the rat-toothed forceps. By pulling on it, the skin is peeled off all around. It is expedient to see that the skin is not pared too thinly, but kept well pulled back with the wound retractors. Then, with a strong pull, the bursal wall is pulled out. After the parts remaining from the anterior bursal wall have been scraped off carefully, the tourniquet is loosened up somewhat, and the larger vessels are ligated. After the lips of the wound have been trimmed, the wound is sutured as described before. Since the hemorrhage rarely stops entirely, it is expedient to use a well-cushioned firm bandage. The horse is either to be tied short, so that he cannot lie down or to be put in a sling to accomplish the same purpose.

REFERENCES

Stang-Wirth, "Tierheilkunde und Tierzucht."
Pfeiffer-Westhues, "Operationskursus."
Möller-Dollar, "Regional Veterinary Surgery."

Lymphatic Dermatitis of Cats

The dermatoses in general, and especially the eczemas proper vary according to their development, their localization, and the nature of the lesions. Incidentally these are the characteristics which actually serve for their classification. Nevertheless, the classification is far from being standard, as clinical observation proves.

Three clinical forms of eczema are commonly described in the cat. They are chronic eczema of the dorsal and lumbar region, moist eczema and arenaceous eczema. Clinical observation prompts us to enlarge upon this too restricted classification, but the lack of constancy of the localization and of the anatomico-pathological character prevents us in general from giving a precise definition of the other forms seen.

Nevertheless, during the past year, we had occasion to observe in the cat four cases of eczema of a very peculiar type, but constant in character. The lesions are always localized in the posterior internal region of the knee and sometimes on the abdomen. The hairless plaques were oval in shape; their dimensions varied from the size of a penny to that of a quarter-dollar. They were raised medallion fashion; the thickness being from 1 to 2mm; the surface moist, hemorrhagic, and well circumscribed. They were sharply outlined from the intact surrounding skin. When scraping this moist surface with a knife, one encountered underneath a whitish, fat-like tissue, resistant and rough. These lesions were always accompanied by a corresponding hardening of the popliteal lymph gland.

The histological section obtained by biopsy showed, especially well, the unusual microscopical lesions.

The epidermis remained only at the periphery of the lesion. The basal layer was disarranged, containing cells of irregular size, their nuclei in caryolysis, and filled in with some lymphocyte-like cells. In the filament-like layer, all the cells, even the deepest ones had a clear, vesicle-like nucleus. The nuclear degeneration was surely more pronounced than in the normal state. The intercellular substance appeared swollen and homogenous, like a sponge. A few lymphocyte-like cells were encountered there. The granulation was well developed; the granulations, normally occurring within the cells, having surrounded the intercellular substance. One found them abundantly in the desquamated layer which follows directly after the granulation. In short, the epidermis, as far as it persisted, presented a picture of cellular degeneration, of sponginess, of keratosis, and of granulocytary infiltration. In many places the epidermis had disappeared, and left the skin nude.

The essential lesion, after all, was not elevated above the level of the skin which presented a very dense granular infiltration; being very thick. The normal elements were literally pushed back, compressed, and formed irregular interstices, separated by deepened, compact masses, the structure of which recalled at first sight that of the lymphoid tissue. There, crowded together, little lymphocyte-like cells, which resembled the average mononuclear cells, a rather great number of histocytes, and some firm, elongated, connective cells were discovered. The capillaries were very numerous; their lumina often filled with lymphocytes; their epithelium unmodified; but often surrounded by a ring of lymphocytes, arranged in several layers. These characteristics of the capillaries seem to indicate that the infiltration was of a hematogenous origin, and not a local cell reaction. In the deeper layers the structure of the subcutaneous cellular tissue unfolded step by step.

The macroscopical and microscopical character of these lesions did not allow their immediate classification into the category of the typical eczemas. The macroscopical appearance suggested an atypical form of ringworm, but the microscopical examination, and the cultures upon Sabouraud medium remained negative. The lymphatic reaction of the skin, the ring of

*Translated from Annales de Médecine Vétérinaire, vol. 81, No. 10.

lymphocytes, the nerve reaction, and the species in question might have lead one to believe in the existence of cutaneous tuberculosis, but the microscopical examination, the cultures, and the guinea pig inoculations remained negative.

On the other hand, the macroscopic appearance of the lesions corresponds almost exactly to a skin affection of the cat, described by Leblois under the term "cutaneous lymphadenitis," and which he attributed to a mechanical irritation from the licking of the eczematous sores. In his opinion, the cutaneous application of a bitter stomachic to restrain the animal from licking should prove sufficient to aid the healing of the lesions.

And indeed, the application of this treatment seems to give some beneficial results. In two animals we were able to observe a certain improvement at the end of two weeks, from the application of tincture of aloes, recommended by Leblois. In reality, this treatment alone is unable to bring about complete healing, much less to cause swift healing. This is easily explained if one considers the organization of the skin lesion. Nevertheless, the simple fact of this improvement is full of interest to us in the sense that it seems to confirm the idea which Leblois put forward of the pathogenesis of this affection. One might consider it as an inflammatory granuloma of mechanical origin, grafted into the preexisting eczematous and pruriginous lesion, producing a lymphatic reaction.

In order to differentiate this ailment well from the lymphadenitis, and to avoid confusion of terms and pathogenesis, we suggest instead of the terms "lymphadenous dermatitis" and "cutaneous lymphadenitis" used by Leblois, the name "lymphatic dermatitis," which seems better adapted to the nature of these lesions.

We were able to obtain complete healing in five weeks by the daily application of a 4% solution of silver nitrate. The recovery is speeded up by irradiation with ultraviolet rays during an exposure of five minutes every two or three days.—*F. Liegeois and P. Terache.*

All Colic in Horses Due to Intestinal Spasm

Today we do not consider colic in horses as a disease entity, but merely as a term, handed down to us from bygone days, for a series of intestinal diseases, differing essentially in their nature, but having in common abdominal pain as a secondary symptom.[1]

Gratzl's studies have shown that the gastro-intestinal diseases characterized by colicky pains have one common etiology, that is, the motility of the gastro-intestinal wall is disturbed by spasms. Complications with secondary factors, however, produce varying new disease forms which have nothing but the colic symptom in common with each other.

In general three groups of colic may be distinguished. The most common form is an abnormal increase in the tonus of the intestinal muscles; this delays peristalsis, contracts the lumen of the intestine, produces congestion, and results in dilatation of the stomach and in tympany. Another form is an abnormal increase in the tonus and rhythm of the peristalsis, causing the so-called nervous colic, spastic colic, rheumatic colic, and enteralgy. The third form consists of intestinal strangulation, due to the dysfunction of peristalsis.

As a rule, errors in management and feeding play merely a contributing rôle in causing colic. Gratzl expresses the opinion that the primary causes are probably organic lesions of the digestive tract. From clinical observations and therapeutical experiments, it was found that the typical variations in colic pain are not caused by over-dilation and expansion of the gastro-intestinal tract in cases of congestion or fermentation of the ingesta, but are due to the violent and varying contractions of the gastrointestinal wall.

An increased tension of the intestinal wall of uniform duration (dilatation or tonic spasm) causes a dull, uniform pain, characterized by apathy, weakness, drooping head, and a peculiar motionlessness when resting,

[1] Gratzl, E. 1936, 1937. Beiträge zum Kolikproblem. (The problem of colic.) *Wiener Tierärztl. Monatsschr.* 23(5):129-138; (9):257-265; (14):417-428; (15):449-455; (20):619-624; 24(2):33-43; (5):131-138; (7):193-203.

or a stiff body position. The severe colic pains caused by volvulus of the intestines are largely due to secondarily-produced, painful contractions of the intestinal wall.

On the basis of clinical observations, the intestinal spasms occurring in colics may be divided as follows: peristalsis accelerated to the point of spasm, and tonic contractions. The former is frequently observed at the beginning of various colic forms, and often terminates in a tonic spasm. Not infrequently, both forms of spasm are observed in the same part of the intestine. While the spasm-like peristalsis exerts an excessively stimulating action upon the movement of the intestinal content, the tonic spasm, on the contrary, causes a retardation, or even a complete stagnation, of the contents.

Rectal examination of apparently healthy horses often reveals a condition of contraction of the intestinal wall, especially at the head of the cecum. Retardation in the movement of the intestinal contents, occasioned thereby, is compensated by a muscular hypertrophy, developing in the course of time.

Clinical observation and post-mortem examination of colicky horses has demonstrated that the various forms of colic are, as a rule, caused by different spasms of the muscles of the digestive tract, while the paralysis of the intestines is usually of secondary importance. The multiplicity of disease forms developing in colic depends upon the degree, the localization, and extent of the spasm. It depends further upon a series of secondary factors, for instance, ingesta, with a tendency of fermentation and swelling, and changes in the composition of the intestinal bacterial flora, coarse fibrous food, slowly digestible food, and disturbances in the secretion and resorption of the intestinal mucosa in fecal congestion.

In cases of intestinal volvulus, many circumstances favor the opinion that the spasm-like peristalsis, and the spastic permanent contraction of the intestinal walls are the immediate cause of the change in position, form, and consistency of the intestine. In the development of the various forms of intestinal volvulus, the secondary accidental causes also play an important rôle.

Thus the various forms of colic all have one common natural cause; i. e., an abnormal increase in the activity of the intestinal muscles. This finding is in direct contradiction to the opinion that the various forms of colic are essentially different diseases.

The forms of colic may be divided into three groups. One group, represented by dilatation, obstipation, and idiopathic bloat, is characterized by stenosis, or occlusion of the pylorus and the intestinal tube, due to the prevailing spasm-like increase in tonus. In the other group belong the so-called nervous colics, spastic colics, rheumatic colics, enteralgy, and catarrhal intestinal convulsions, characterized by excessively rapid evacuation of the intestines, resulting from the excessively stimulated rhythm of the peristalsis. The third comprises volvulus and torsion of individual sections of the intestines, caused by spasm-like intestinal movements.

Very often individual colic cases cannot be placed, either clinically or anatomically, into any specific colic group. Frequently during the course of the disease one is able to diagnose simultaneously differing conditions in the same animal. Thus, the clinical and anatomical pictures, too, are in opposition to the view that the various forms of colic are diseases differing essentially in their nature.

Clinical experiments on healthy horses and on those suffering from colic, carried out with different drugs have demonstrated that in colicky horses the action of the parasympathicus predominates. The fibers of the parasympathicus reaching the intestines are in a state of excitation and possess an increased excitability. This is apparent in the differing behavior of healthy horses and colicky horses towards drugs which stimulate the parasympathicus.

Thus, the predominance of the vagus tonus must be considered as being the cause of the abnormal rise in muscular activity of the intestine occurring in colics.

It has been shown that chronic and

acute inflammatory, partly degenerative and atrophic lesions of the sympathic ventral ganglion and nervous fibres of the plexus mesentericus exist. Since the sympathic and parasympathic nerves leading to the gastrointestinal tract exert a stimulating as well as a restraining action upon the motility of the intestine, one is justified in concluding that the nerve lesions mentioned are capable of exerting a disturbing influence upon the motory functions of the intestinal wall.

Various authors, over a period of many years, have observed that colics are likely to occur following exposure to cold (internally or externally), when proper care is lacking, when the animals are overexerted, and especially before weather changes. If one compares with it the so-called foretelling of the weather by persons with nerve lesions (rheumatism, neuritis), it is obvious that the nerve lesions caused by the verminous aneurysm, which seems to be of rather frequent occurrence, do at times produce a hypersensitivity toward the stimuli mentioned.

The action of these stimuli, suddenly disturbs the equilibrium between the sympathicus and parasympathicus tonus, which had become liable due to changes in the nerve. Thus, it is made possible that abnormal disturbances of motility may occur in the digestive tract. By their difference in form, in localization, and by the addition of a series of secondary factors, they are able to produce the various forms of colic.

The tonic spasms observed in apparently healthy horses may be considered as an additional consequence of the changes, mostly chronic, in the intestinal nerves. They are caused by a hypertrophy of the muscles of that part of the intestine situated near the stomach. In cases of sudden change of food, or when nearly indigestible food (straw) is ingested, a slowly developing impaction of the contents is likely to occur. When these tonic spasms increase in intensity, they give rise to habitual impaction, especially in the cecum. This impaction is difficult to influence therapeutically, and when relieved will reoccur as feeding is resumed.

This theory concerning the etiology of colic is in contradistinction to the view that the individual colic forms are essentially and naturally different diseases of the digestive tract, which opinion is founded upon the great difference found in post-mortem examination, and partly upon clinical (rectal) exploration.

Regarding the prevalence of vagus stimulation prevailing in cases of colic, drugs which stimulate the tonus of the intestinal wall are to be avoided in treatment. Members of the pilocarpin group, and also barium salts usually aggravate the symptoms of the disease and are likely to produce rupture and volvulus.

At present, lentin,* belonging to the pilocarpin group, is the most satisfactory remedy. Drugs possessing a sedative action upon the intestine would really be the best agent, but in most cases they go too far in their action, producing an atony of the intestine lasting for some time. Morphine is to be avoided in the treatment of colic. The best antispastic drug for colic, at present, is atropin, although it possesses a series of undesirable qualities. The therapeutist is still looking for a sedative which does not paralyze peristalsis.

The most effective form of hydrotherapy in colic is cold lavage of the stomach, which is not difficult to perform if done correctly. The value of gastric lavage is not only to give relief to the stomach through the evacuation of gases, fluids, and solid particles in cases of dilatation, but, still more important, to relieve the spasms and stimulate peristalsis throughout the entire digestive tract. From experience gathered at the medical clinic of the veterinary college at Vienna in the treatment of dilatations of the stomach, of catarrhal spasms of the intestines, of tympanites, and the lighter forms of impaction, cold lavage of the stomach is generally the most successful treatment, and is superior to any drug therapy.**

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*See Burgett, M. V., and Bardens, J. W. 1937. Treatment of impaction in the horse with Lentin, the new rapid acting cathartic. *Jour. Amer. Vet. Med. Asso.* 43(5): 646-655.

**Compare also abstract by D. H. Udall in *The Cornell Veterinarian*, vol. 26, pp. 131-137, "Treatment of impaction of the cecum of the horse."

Veterinary Service in Venezuela

By CARLOS J. CARDONA,
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SINCE I have been working with the Technical Division under the Ministry of Agriculture of Venezuela, I have wished to bring to the consideration of the veterinarians of the United States facts about several diseases of livestock common to Venezuela. Doubtless many veterinarians in the U. S. A. must be eager to know more of the ailments attacking both the bovine and the equine species. As far as I know, the instruction given students in American schools concerning tropical diseases is far below that which should in all reason be given. I know from personal experience that what is taught veterinary helpers here in Venezuela in every branch of tropical disease is above the level of what is taught the veterinary student in the average American veterinary college.

I shall begin by saying that Venezuela occupies an area of twice the size of Texas and has a population of more than three millions. Until 1935 nothing was known of the diseases that for years have attacked livestock. Today a campaign is being fostered by the Ministry of Agriculture to find methods of eradicating disease. The Livestock and Technical Divisions are developing plans to bring to this end or to bring disease under measurable control. The Division employs a number of veterinarians from Argentina, Chile, Czechoslovakia and Cuba. They travel about the country and investigate disease and zootechnics. With them goes a trained helper. After only a two-year course in infectious diseases, these veterinary helpers can handle a microscope efficiently and can perform a number of surgical operations and do a good job in post-mortem work.

Piroplasmosis—Anaplasmosis

Piroplasmosis in cattle is met with occasionally in conjunction with anaplasmosis. Both diseases, alone, are very common, being usually more virulent in imported cattle. In school I never heard of a reliable, efficient treatment; here we use a good German product known as "acaprina," made by Bayer. It is given intravenously in doses of 1 cc per 100 kgm. Tripaflavine administered in 50cc doses intravenously also gives excellent results.

Mal de Cadersas—"Hip Disease"

This disease is caused by the *Tripanosma venezuelaiense,* and is very common among horses and donkeys. Early treatment with Bayer 205, naganol, has saved many an otherwise doomed animal. Antimosan has also saved many horses, especially when given during the first stages of the disease.

Strangles

Hermosa, as strangles is called, is very common here and is usually treated as in America. Preventive practices are commonly used.

Canine Distemper

Peste en los perros, or canine pest extends even to the most remote corners of the plains. This disease attacks dogs and foxes. The mortality is very high, and as distances are so great and traveling conditions bad, treatment is usually futile.

Rabies in Dogs and Foxes

Rabies is a very common condition among the carnivora in elevated regions. The disease is said to be transmitted from dogs to foxes. Vaccination against rabies is not practiced in Venezuela. There are no national, state, or municipal laws regulating the disease. Human cases of rabies are common.

"Borrachera," or Drunkenness

This condition affects only cattle in good flesh pasturing on the banks of the Orinoco river. Beginning May 30th every year the river level begins to fall, the areas formerly covered by water become dry (November) and are covered with grass. This is the time when cases of borrachera begin to show up. Cattle thrive on the grass left by the receding waters. The disease becomes noticeable as soon as the cattle are moved into a new lot or pasture. If they happen to stampede, the losses are increased tremendously. Cattle

may show not the slightest symptoms of the disease, all be pasturing quietly when a slight excitement will start them trembling. The extremities and head are moved constantly. They wabble and struggle, and in a few minutes fall dead. Some patients are very excited and run great distances before dropping. The losses are great from November or December until May. It has been impossible to discover any microscopic evidence that sheds light on the disease. A preventive consists of putting the cattle on a hunger diet for two days before they are moved either to the corral or along the plains to market. Such abstinence from the deadly grass saves them from sure death, even if they run wild or are excited all the way along the plains from the overflowed pastures.

I believe that this condition is an alimentary toxicosis affecting cattle and that the only means of decreasing its high mortality is to prevent the cattle from eating the grass left after the water has receded.

Post-mortem lesions show a congestion of the meninges and brain, congestion of the lungs and pleura. Petechia on the intestinal mucosa is occasionally seen. The most remarkable lesion is seen on the tissues lining the rumen. Right after death these tissues are very soft, appearing as if boiled. The villi are easily peeled off at the slightest touch with the fingers. This condition occurs only on the banks of the Orinoco river and its tributaries, never along interior rivers and creeks.

Tropical Sporotrichosis

An indolent ulcer called "espundia," affects only horses pasturing on areas flooded by the waters of the Orinoco river, and also on the lowlands watered by the river. The boils are found on the extremities, ventral part of the abdomen, brachial region, scrotum, penis, thighs, and mammary region of the mares. At first the boils are insignificant; later they invade the subcutis and musculature, forming hard nodules. The area increases in size and thickness, usually forming an abscess that may rupture spontaneously after two or three weeks. After the abscess discharges, there remains in the center a hard, whitish growth. The base of the ulcer and the periphery contain a hard mass of connective tissue with numerous pus foci that reach the base of the ulcer and have a diameter of 5 to 8mm.

Many horses are attacked every season, large numbers dying if not properly treated.

Peste Seca

Peste seca or dry pest attacks horses in any season; poor horses as well as healthy ones. They lose hair in the first stages, and begin losing weight two or three weeks later; continuing to lose flesh until cachexia is present. There is no pyrexia, and the patient maintains a good appetite until the end. The disease usually runs a chronic course, the patient dying in two to four months. Several that I have examined showed dental irregularities; many also suffer from chronic parasitism.

The main post-mortem lesion is in the spinal cord, which becomes soft and sometimes degenerates to liquifaction.

Anthrax

This ancient plague of domesticated animals is very common in Venezuela. It is easily diagnosed, bacteriologically, by the reliable method known as "olt." Eradication and preventive measures are practiced.

Hog Cholera

This disease is common to isolated areas, but is not generally distributed over the republic.

Azoturia

Bleeding is the common treatment for azoturia. It is used even by graduate veterinarians. I have preached in vain against the dangers of such a practice.

Dourine

Dourine is occasionally observed.

Summary

I hope this brief discussion will suffice to give the veterinarians of the United States an idea of the principal diseases of animals common to Venezuela. Needless to say, there remains a wide field to be explored. I am anxious to help anyone wishing information on tropical diseases.

I wish veterinary students might better appreciate the value of a clinical training and a thorough preparation in laboratory technic, stressing both bacteriology and pathology.

Summary of Infectious Diseases at World Poultry Congress

The following is an extract (Deut. tier. Woch.) of a paper read by Meissner and Berge before the Sixth World's Poultry Congress at Leipsic, Germany, July, 1936.

Pasteurellosis.—In addition to the usual septicemic type, pasteurellosis can become localized in the wattles. Carriers can be detected by sero-agglutination. Manniger, who believes the disease is not contagious, attributes outbreaks to lowered resistance brought about by exposure to cold, faulty alimentation, fatigue of shipping, worm parasites, and the like.

Prevention is accomplished principally by passive immunization with hyperimmune bovine serum, but longer protection can be obtained with serum used in conjunction with avirulent organisms. Vaccines made with formalin (1:1,000) or gentian violet (1:2,000) are widely employed in Russia. Vaccines attentuated with saponine generally cause mortal infection.

Antiseptics in the drinking water (iron sulphate, creolin) have preventive properties.

Tuberculosis.—The intradermal (wattle) tuberculin test has real diagnostic value, but inasmuch as complete change of flocks every second year, together with the removal of the poor layers and good hygiene, eliminates the disease, the test loses its importance.

Diphtheritic variola.—Vaccination used with hygienic measures is efficacious. Chicken pox virus passed through pigeons or rabbits is preferable. While the vaccinal lesions of original pigeon virus cure most rapidly, the immunity produced is less durable.

Annual vaccination should be practiced on all fowl over three months old. It should be done preferably in August or September.

Urotropin (methenamine) in 10% solution, given subcutaneously or intravenously, possesses curative properties. "Liquid sulphur," 2 to 3%, is prophylactic (Richter).

Infectious laryngotracheitis.—Widely existent in the United States, is distinguished from chicken pox (diphtheritic variola) by the presence of an exudate in the trachea and bronchial tract. The pathogenic agent is an ultramicroscopic virus which may persist in cured subjects for sixteen months. The prophylaxis rests upon hygienic measures and intracloacal vaccination.

Contagious coryza.—The etiology of the affection, observed in chicken and occasionally in pigeons, is still disputed. De Blieck incriminates hemoglobinophile bacterium whose intranasal application produces the disease. The prophylaxis consists of rational housing (cleanliness, ventilation), and alimentation (calcium supplements).

Fumigations with creosote and turpentine 1:3, and antiseptics sprayed over the flocks are the convenient treatments. Individual treatment consists of bucco-pharyngeal swabbings with iodized glycerine and nasal sprays.

Leucosis.—Recent researches enable one to distinguish two forms: One is non-transmissible, affecting the lymphatic system exclusively; the other is transmissible, attacking the marrow, and is subdivisable into erythroleucosis (presence of erythroblasts in the blood) and myelosis (multiplication of myeloblasts). It is considered due to a pathogenic agent of a diastatic nature, formed by leucocytes in the process of proliferation. The disease, therefore, resembles transmissible neoplasms (sarcoma of Rous).

Repeated injections of the bile of affected fowls confers a slight immunity (Wakamatsu). The use of lead (Zedik), of quinine plasmochin and rhodoquin (Oberling and Guérin), appear to give favorable results.

Neurolymphomatosis.—Everyone is in accord as to its intestinal origin because of the constant presence of pronounced duodenitis (thick, velvety mucosa covered with white exudates). Pappenheimer was the first to point out the mistake of classing this disease with leucosis and to propose the

*Free translation from *Annales de Médecine Vétérinaire*, January, 1937.

name, "neurolymphomatosis." The cellular infiltration of the nerve trunks is supposed to be due to the stimulation of the lymphatic system.

Prophylaxis is purely hygienic, destroying the sick and systemic disinfections and cleaning.

Pullorum disease.—The control of this disease necessitates the elimination of the carriers by sero-diagnostic methods, good hygiene, and the selection of resistant breeds, together with good organization among breeders working under public authorities.

Bacteria of the paratypho-enteridic group.—The bacillus of Breslau (Aertrycke) often contaminates eggs, especially of ducks, and also of pigeons and geese. Such eggs can produce alimentary intoxications in man by the consumption of mayonnaise dressings, puddings and the meat of geese. Moreover, web-footed fowl and pigeons are a source of infection for horses, cattle and swine (Lerche). Hygienic control, especially of ducks, imposes itself.

Contagious tremors of chicks.—This disease was signalized by Jones in the United States in 1930. It is characterized by trembling of the head and neck, and sometimes the tail, with ataxia. There is no fever nor alteration of the blood picture. The lesions are found in the brain and spinal cord. Its mode of transmission is not known. Mortality varies from 0 to 65%.

* * * *

Mechanism of Oxygenation

Anemia is a reduction below normal of the capacity of the blood to transport the oxygen necessary for all animal life. The body tissues must be supplied with many times as much oxygen as can be carried in physical solution in the plasma. The hemoglobin normally present increases one hundred times the power of the blood to transport oxygen by carrying it in chemical combination. This amount of hemoglobin in solution in the circulating blood would greatly increase the osmotic pressure of the plasma beyond that of the surrounding tissues and so dehydrate the tissues. Hemoglobin in a red cell is outside the plasma, does not affect the osmotic pressure, and yet functions efficiently as an oxygen carrier since absorption and release of oxygen is as efficient as if the hemoglobin were in solution in the plasma. The red cell is thus simply a container for the necessary hemoglobin and functions as a cup on an endless-chain conveyor. It is normally filled with hemoglobin and is constantly making round trips from the lungs to the tissues.—*R. L. Haden* in *Jour. of Lab. and Clin. Med.*

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A Choice of Explanations

In falling, the cat always lands on her legs. The reason for this has been revealed by the slow motion camera. When the cat is held by her legs in an upside down position, and then released to fall a certain distance, she begins immediately to turn her tail in a rotary motion like a crank. In consequence of this motion of the tail, the body revolves in an opposite direction. The crank-like motion is continued until the body regains normal position, that is, until the legs are directed downward. As soon as this position is regained, the rotation of the tail ceases, and the cat lands on her feet.

The cat is able, when she falls, to turn in the air and land squarely on her feet.

The rotation in the air will occur to one side or the other, depending on the animals initial position. If she is held with her back down, her legs inclining to the right, she will turn to that side; if the legs originally point more to the left, the rotation will be in that direction. Motion pictures show that the animal first contracts her forelegs and then turns her forepart around. This makes the hind part turn in opposite direction, but to a less extent. Then she contracts her hind legs, extends the forelegs, and gives the hind part a turn. This turns the forelegs a little again in the opposite direction, but again slightly. The cat can turn herself through any angle by continuing contractions of this kind.—*G. I. Gates.* "The Modern Cat: Her Mind and Manners."

Chemical Medication*

By C. D. FOLSE,
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About twelve years ago, I published these statements: "The basis of medical science is essentially chemical. Though periodically, systems and medicaments flourish in a way to indicate possible elimination of the chemical treatment of disease, the reaction invariably returns chemical medicine to a stronger position than before. It is logical to predict that ultimately perfection in medical skill will be worked out along chemical lines. The evolution of the several arsenical preparations for the treatment of syphilis is an illustration of the possibilities in other diseases.

"It is not intended to minimize the value of certain animal products which at present have a definite usefulness and for which there is no substitute, chemical or otherwise. Anti-hog cholera serum and blackleg aggressin are examples. The essential idea is to keep in mind the possibilities of chemistry and strive to develop its potential powers, which are as yet hidden in our ignorance of the vital chemical reactions."

I review these statements now because I still believe them. Several discoveries of the past decade proclaim their fulfilment. Now as then, I want to stimulate you as practitioners to do your all-important part in bringing them to a full realization. I have a vision of the veterinary practice of the future in which animal diseases will be approached from the chemical etiological angle. Then diagnosis, eradication, control and treatment will be centered upon the logical basis, which is purely chemical.

A review of the accomplishments of our profession in relation to the control of transmissible diseases demonstrates that most satisfactory progress has been made by means of sanitation and other chemical procedures. Notable among these are Texas fever and tuberculosis. The inefficiency of disease control by means of biologic agents is illustrated by hog cholera. Anti-hog cholera serum is nearly perfect as a biologic control agent, but after many years of intensive effort, hog cholera is still as prevalent and as economically costly as ever. If the money, time and energy spent in hog cholera control had been devoted to chemical research, who knows what might have been the result. The recent creation of crystal violet virus suggests progress toward a less expensive and less wasteful solution of the hog cholera problem.

Organisms responsible for disease are often indigenous to their host and become pathogenic only when favorable conditions develop in the tissues of the host. Practitioners know that abortions in dairy cattle can often be controlled by including a mineral supplement in the ration. It has been learned that anaplasmosis may be checked by attention to the diet, and rickets may be prevented by supplying sufficient vitamin D. What may be spoken of as non-virus canine distemper—a bacterial infection—is completely cotrolled by adequate nutrition.

Concrete examples of chemical control of disease, some infectious, are many. As a basis for contemplation, a few are suggested. The remarkable effect of mineral supplements is too well known to need comment. The abuses of commercialization of this product have caused some confusion, but the virtue of the idea is established. An ideal illustration of the value of minerals is offered in the prevention of bony diseases in race horses and harness horses by supplying a mineral supplement to the mare during pregnancy and during the suckling period.

The handling of necrotic enteritis in swine by medication with solutions of the alkaline powders, is an outstanding attainment, because necrotic enteritis is possibly the most devastating, economically, of all swine diseases.

The administration of willow bark and arsenic in fistula of the withers, poll evil and other suppurations of a chronic nature,

*Elaboration of an impromptu talk at a meeting of the McLean County Veterinary Medical Association at Bloomington, Ill. Sept. 26, 1931.

has solved a very annoying problem. The expensive and unsatisfactory surgical procedure is now limited to the few cases which do not respond to chemical treatment.

The employment of sodium iodide in capillary hemorrhage; of sodium perborate in putrefaction and infections of placental membranes; of the flavines in malignant edema and calcium salts in milk fever and other disturbances of the calcium balance, are all familiar chemical treatments.

Study of chemical agents discloses that success in their use requires intelligent consideration of the drugs themselves and of the methods employed in their administration. Much of the required information is not clearly stated in the textbooks. It may be helpful to call attention to some of this information concerning chemical agents.

A detail often overlooked is the degree of irritation a chemical may produce when brought into contact with the tissues. Copper sulphate and sodium hydroxide are outstanding examples of valuable caustic chemicals which can be utilized to decided advantage if administered properly. There is perhaps no more effective germicide and vermicide than copper sulphate. It is active in solutions as dilute as 1 to 5,000. When diluted to 1 to 1,000 (1 pound in 120 gallons), copper sulphate ceases to be harmfully irritating to the mucous membranes of the digestive tract. In this dilution, however, it is capable of destroying, or at least rendering inactive, most of the pathogenic flora and animal parasites encountered in the digestive lumen of domestic animals. A 1 to 1,000 solution of copper sulphate can be depended upon to control most of the intestinal infections of all animals and the protozoa of all animals. When combined with sodium hydroxide, it is most effective against tape worms in poultry and sheep.

Toxicity is another factor involved in chemical medication. Toxicity varies, of course, according to the size of the dose and the species of animal. Strychnine, for example, is very toxic to dogs and practically nontoxic to chickens. Caustic agents in concentration destroy tissue, but are often harmless in proper dilution. Calcium salts cause sloughing where injected into the tissues, but produce no harmful effect in the blood stream. Arsenic in the form of arsenous acid can be administered in huge doses orally to dogs, but is deadly to man. Arsenic is a valuable agent for correcting stubborn skin lesions, but its toxicity in the human family has caused it to be avoided largely in canine practice. Thymol is toxic when administered in the presence of fats.

Dosage is always an important element of medication both as to sufficiency and as to manner of administration. Calcium gluconate solution offers an example of the possibility of insufficient or over-dosage. The dosage of this product cannot be stated accurately on the label, because too many factors are involved in the behavior of calcium in the tissues. The intravenous dose for a parturient paresis case, for instance, may vary from 90cc or less to 500cc or more. Unfavorable results from calcium injections are doubtless often due to failure to recognize this variability of dosage. There are certain farily definite reactions to calcium injections which may be utilized to determine a fairly accurate dosage. The effect upon the heart should be manifest in a fuller, stronger pulse; the effect on voluntary muscles should appear in a twitching of the superficial muscles, and the effect on the voluntary muscles and reflex nerve endings, in an emptying of the bladder and bowel.

In some cases best results are obtained by using small doses over a period of time. This is true of any medication looking to the control of intestinal parasites, especially tape worms. In my opinion, one dose (tablet, capsule, or liquid) is very objectionable. In round worms, one dose medication expells only the adult worms, leaving the embryos present and those to be picked up later, to produce reinfestation. In tape worms, the head is embedded in the mucosa and surrounded with mucus; hence, one-dose medication expels the body segments, leaving the growing head intact. A regular daily dosing over a period of ten days, repeated every six to eight weeks is the logical method of treatment.

While the importance of vitamins has

probably been greatly overrated, these substances do have an influence on metabolism which affects medication. The calcium deficiency exhibited in rickets is an illustration of vitamin influence. Unless vitamin D is present in the ration, calcium known to be present in abundance is not utilized in the bony structures, and rickets results.

The endocrine glands also play so great a part in normal body functions that disturbance of one or more of these structures may render otherwise correct medication inoperative, or even harmful. The relation of the thyroid gland to iodine utilization is an example.

Incompatibilities of chemicals must be considered. The use of rumen compound tablets with epsom salts is an example. The sulphate in the epsom salts reacts with the barium chloride to form inactive barium sulphate. Experience sometimes shows that textbook teachings are incorrect, as in the case of aconite and digitalis. Actual tests show that aconite and digitalis employed simultaneously, do not neutralize each other physiologically, but that a stronger, more forceful heart action is produced. In relation to incompatibilities, it is well to remember that magnesium sulphate cannot be used with either calcium gluconate or sulfanilamide.

Preparation of the patient must be taken into account in some instances. In the use of taeniacides, it is advisable to prepare the patient by flushing the intestinal lumen with magnesium sulphate or an alkaline agent to remove mucus adhering to the mucosa and covering the heads of the attached tape worms. Contrary to the general rule, anthelmintic medication in poultry should be followed by a full feeding to carry the medication out of the crop as quickly as possible.

To handle chemical medication properly, each chemical must be considered as an individual and in relation to other drugs when it is combined in medication. To illustrate, let us take three common drugs.

Aloes is supposed to derive its action from its glucocide, aloin. Aloin, under proper conditions, produces in the intestines a substance called anthracine, the substance which induces the action of the bowel. Extensive tests have established the fact that pure, unadulterated aloin is inert, capable of no action whatever. The practitioner knows that aloin often fails to act as expected, and this failure is due to the fact that the aloin is pure. Aloes contains a resin which is essential to convert the aloin in the bowel. In the water extraction process this resin, being insoluble in water, is left in the aloes residue. Fortunately, pure aloin can be made active by the addition of either iron sulphate or an alkaline substance, as sodium bicarbonate.

Arecoline has lost a great deal of its popularity in recent years, due, I believe, to lack of a clear understanding of its dosage. In proper dosage it is a powerful stimulant of the musculature of the rumen in cattle and of the intestines of cattle and horses. In too large dosage, there is over-stimulation, to the point of paralysis, and this is probably the cause of much of the unsatisfactory actions the practitioners have experienced. Undisputable tests demonstrate that ⅛ grain administered subcutaneously is as large dose as should ever be given hypodermically to cattle. In the horse, ⅛ to ¼ grain, reinforced with ⅛ grain of strychnine is most likely to give satisfactory action. Any action which may result will be evidenced by movement in the rumen of cattle and in the bowel of horses in 15 minutes. If such action is not noted, the dose may be repeated. In dogs, the oral dose should not exceed 0.1 gr. in medication for tapeworm. If no action results in 15 minutes, repeat.

Oil of chenopodium is perhaps as good an anthelmintic against ascarids in swine as is obtainable. The chief objection to it is the tendency of chenopodium to produce severe irritation of the gastric and intestinal mucosa. In an effort to overcome the irritation, chenopodium is often mixed with castor oil or some other vegetable oil. This practice is undesirable, because the vegetable oils decrease the effectiveness of the chenopodium. A much better diluent is an inert or adjunctive powder. Chenopodium medication should be followed in six to eight hours by a purgative agent. Here, too, the oils should be avoided. Salines or aloes is more desirable.

As before stated, copper sulphate is a highly effective germicide and ascariacide. Because the textbooks are in error concerning the internal dosage of copper sulphate, practitioners do not employ the agent as much as its value warrants. Administered in solutions of 1 to 1,000 or weaker, there is no danger of overdosing or of producing any irritation of the musoca. This solution of copper sulphate is deadly to pathogenic flora and animal parasites alike if medication is continued over a period of time.

Magnesium sulphate has a large field in veterinary practice as a sedative, and it is unfortunate that the dosage of this product was so grossly understated when it first came into use. For colics of the spasmodic type, for dystokia, for straining in diarrheas, for tetanus and in other conditons where a prolonged sedative action is desired, epsom salts in 20% solution is very useful. The minimum dose for horses and cattle is 30cc, and as much as 1,000cc may be used. The injection should always be made into the muscle tissue, not more than 20cc being injected at one point. Magnesium sulphate solution should be employed in dogs with extreme caution.

I have offered this brief sketch of chemical medication to you as practitioners, because I recognize that you are the essential element in the development of this important part of veterinary medicine. Laboratory workers may check and elaborate your findings, but in the end you are the deciding factor. I urge that you abandon the tendency to accept commercial literature and the label on the bottle as gospel, that you may give the profession your thoughtful aid in solving the many problems which now seem hopeless.

I repeat that I visualize a veterinary practice in which the practitioner will scrutinize carefully each case, observe and record all the details, both of diagnosis and treatment. He will refuse to guess at anything and will not be satisfied with medication which does not produce the reaction he expects. He will accept the claims of others only after he has demonstrated their correctness himself. With such a vision realized, I am sure we may have a veterinary profession which will be so indispensable to the livestock industry that its value will be recognized. We will then be in a position to plan and enforce scientific control and eradication measures.

Operation for Capped Elbow

Indication.—The simple incision seldom gives satisfactory results. Extirpation of the cystic bursa is recommended in all cases of well developed serous, fibrous or purulent inflammation of the bursa.

Instruments.—The surgical equipment needed consists of razor, luer syringe, scalpel, probe-pointed scalpel, scissors, rat-tooth forceps, clamp forceps, tenaculum forceps, tampon, silk, suture material.

Narcosis.—The skin in the area of operation is to be infiltrated with a suitable anesthetic. If the horse appears to be unmanageable, chloral narcosis may be advisable.

Technique.—After the necessary preoperative preparation, the horse is cast. The operative area is shaved, painted with tincture of iodine, and anesthetized. With slightly arched downward strokes, meeting at top and bottom, the skin is separated, leaving a spindle-shaped piece of skin on the bursa to be peeled out. By dissecting toward the base of the cystic bursa, the skin is lifted off all around. Severely bleeding vessels have to be ligated. Care is to be taken that the skin flaps remain at least 1cm wide. As soon as the base of the hygroma is reached, the capsule is lifted up with the left hand and severed from the cord-like connection. In order to facilitate healing, all bleeding vessels have to be ligated. The lips of the wound are now brought in apposition to form a ridge 1cm high.

The suture is started from the upper angle and carried down with continuous stitches, leaving the lower angle open for drainage. A quilled suture is then put in place to relieve tension. For about 10 to 14 days the horse should be tied up short, to facilitate the healing process.

REFERENCES

Stang-Wirth, "Tierheilkunde und Tierzucht.'
Pfeiffer-Westhues, "Operationskursus."
Möller-Dollar, "Regional Veterinary Surgery."

Drugs Used in Veterinary Medicine

VII. BARBITURIC ACID DERIVATIVES

By VICTOR LEWITUS, New York City

Historical Sketch

ORDINARILY, the animal mechanism requires rest following a period of activity. When this period of quiescence, whether actual sleep or another form of inactivity, is seriously hampered because of irritability of a general character or following disease, there is an immediate effect upon the animal functions. To combat such abnormal states, the practitioner has certain sedatives at his disposal, copied in many cases from nature's pattern, but in other instances free of certain natural disadvantages. Wishing to break away from the habit-forming character of the poppy (opium), early chemists began to carry on various syntheses. Liebreich, in 1869, first produced chloral, which soon contributed to the popularization of "knock-out drops." This forerunner of the hypnotics was found unsafe, and so chemists sought farther for the solution of the difficult problem. They studied various combinations with urea and discovered that in conjunction with alcohol a product known as urethane gave good hypnotic results, even though the action was not so powerful as that of opium or chloral. Then came the production of sulphonal and trional, which brought them a step nearer the goal. In turn, these products gave way to a most remarkable series of drugs, powerful hypnotics discovered by Nebelthau in 1898. These artificial substances have their beginning in a combination of derivatives of malonic acid ($CH_2.(COOH)_2$) with urea ($CO.(NH_2)_2$). The most important of these drugs is barbital, established in 1904 through the work of Fischer and Von Mering. Barbital substance served as a basis for the other barbituric acid derivatives which followed in the search for even greater improvement. One of these investigations resulted in the development of luminal or phenobarbital, and another, dial. Further progress with the barbiturates came in 1920 when the newer alcohols were combined into more barbituric acid derivatives. As a result, we now have ipral, neonal, amytal, and others. We have now over 60 barbituric acid derivatives available in medicine, each differing a bit from its close relatives. There is no doubt that in the field of hypnotic drugs, synthetic chemistry has earned a place for itself as a modern "miracle man" for within the span of only 30 odd years has this field of medicine achieved outstanding success. Nor have the possibilities for improvement been exhausted!

Compounds and Properties

Official. — Barbital (veronal), soluble barbital (sodium barbital), phenobarbital (luminal), soluble phenobarbital (luminal sodium), elixir barbital, elixir phenobarbital.

Unofficial.—Dial, neonal, amytal, sodium amytal, seconal, ipral, phanodorn, pentobarbital sodium (nembutal), alurate, nostal, ortal sodium, sandoptal, pentothal sodium, allonal.

Barbital (veronal) is chemically diethylbarbituric acid or diethylmalonylurea ($(C_2H_5)2\ C{\cdot}CO{\cdot}NH{\cdot}CO{\cdot}NH{\cdot}CO$. It occurs as colorless crystals or a white powder, having no odor, and a slightly bitter taste. It is readily soluble in alcohol, but only slightly so in water (1:130). This drug, the forerunner of the barbituric acid series listed in the foregoing, is an effective sedative and hypnotic. It is also an anticonvulsive and is used in cases of chorea, general restlessness and in allaying nervousness, as a pre-operative step.

It augments the action of analgesic agents, especially in the relief of neuralgic pain. The margin of safety between the therapeutic and toxic dose is wider than that of chloral, which it exceeds in hypnotic power. Barbital is absorbed quickly, takes effect within half an hour and lasts from four to eight hours.

Since the drug is excreted slowly, cumulative toxic effects may be induced. Habituation occurs in animals, and doses must usually be stepped up accordingly, although

with great caution. It may be used as an antidote in case of poisoning by strychnine, picrotoxin, cocaine and procaine hydrochloride.

Soluble barbital (sodium barbital) is chemically sodium diethylbarbiturate, or sodium diethylmalonylurea $(C_2H_5)_2C.CO.N:CONaNH\cdot CO$. It is a white, crystalline, odorless powder, having an unpleasant, bitter taste. It is freely soluble in water and slightly so in alcohol. Its other properties are the same as those of barbital, except that its greater solubility facilitates more rapid absorption.

Phenobarbital (luminal) is chemically phenylethylbarbituric acid or phenylethylmalonylurea. $C_6H_5\cdot C_2H_5\cdot C.CO.NH.CO.NH.CO$. It occurs as fine, white, shiny crystals or powder, odorless, slightly bitter, slightly soluble in water, but soluble in alcohol, ether, chloroform and alkaline solutions. It is hypnotic and sedative, like barbital, but it also has a pronounced sedative action on respiration and in overdose kills by respiratory paralysis. It is partially eliminated in the urine and partially decomposed in the body. It produces urticaria at times. The drug is used to reduce nervous irritability and convulsive conditions.

Soluble phenobarbital is the sodium salt of this drug. It is readily soluble, and for this reason is indicated where prompt action requires intravenous administration. (The elixir of barbital and phenobarbital are diluted preparations not intended for veterinary use.)

Pentobarbital-Sodium (*nembutal*) *is* chemically sodium ethyl (l-methylbutyl) barbiturate or sodium (methylpropyl carbinyl) barbiturate. $NaC_2H_5(CH_3CH_2CH_2(CH_3)CH)CCONHCO:NCO$. It occurs as a white crystalline, odorless powder, with a slightly bitter taste; very soluble in water, freely so in alcohol, but insoluble in ether; it is alkaline in aqueous solutions. This drug resembles soluble barbital, but is more active, and therefore smaller doses must be given. The duration of its action is relatively brief, an advantage if large doses are used. Used pre-operatively, it must be administered with caution. It may be given orally or rectally, but the intravenous method is often employed for small animals. Intravenous methods require caution. Since it is of the short acting group, nembutal or pentobarbital is now being used as a general anesthetic in small animal practice. The age, weight and general health of each animal must be considered in estimating the dose. Young and undernourished animals require slightly less per pound than older, heavier animals. The average dose, regardless of the method of administration, is 0.2 gr. per pound of body weight, or one grain for each five pounds. It is seldom necessary to give large dogs more than a 10-grain dose. In intravenous or intraperitoneal injections, anesthesia usually results in from three to ten minutes and usually lasts from an hour to an hour and a half. There is no excitement, nausea or vomiting, the animal remaining quiet for several hours after the operation. The makers of nembutal recommend that in cases of incomplete anesthesia (that is, if anesthesia does not take place in twenty minutes) another injection should not be attempted, since it would probably be fatal. In such a case, it is best to follow with ether inhalations. Quite possibly in a material percentage of such cases a subcutaneous dose has been given instead of intravenous dose as was intended and thus absorption was slow and anesthesia incomplete. For intravenous use, the external saphenous vein should be employed. The injection should be made *very slowly*, and anesthesia will have been produced as soon as injection has been completed, or rather the injection is discontinued when anesthesia is complete. The degree of anesthesia can be controlled by observing the pupillary reaction. At the onset of anesthesia, the pupil dilates, and as the injection continues, begins to contract. Once the pupil contracts to about ¼ or normal size, the injection should cease, since safe surgical anesthesia is then indicated. When used intraperitoneally, the injection should be made on the posterior surface of the abdomen on either side of the median line. By using a course needle carefully, the puncture of a distended bladder or intestine will be avoided. It is best to elevate the hind parts, allowing the internal organs to fall forward,

thus facilitating proper injection. When given orally, best results are obtained after a 12-hour period of starvation. Thus absorption will take place promptly and anesthesia soon follow. If the animal's stomach is not empty, excitement will undoubtedly follow upon oral administration. Many veterinarians find an ideal combination in morphine and nembutal used as follows: The morphine is injected intravenously at the rate of one-half to one grain, according to size of the animals. The stomach will thus be emptied and a period depression follow. Thirty minutes after giving the morphine, nembutal at the rate of 0.1 gr. per pound live weight, is injected, either intravenously or intraperitoneally. Nembutal has been suggested for eclampsia in bitches, ewes and sows at the rate of 1/5 gr. per pound body weight. In sheep and swine the same dosage is prescribed, using the intraperitoneal or intravenous route. In sheep the jugular vein should be used, and in swine a vein in the ear is suggested. When large animals do not respond to ordinary sedatives in case of colic, azoturia or tetanus, nembutal may be used intravenously in a dose of 15 to 35 grains. In the early stages of strychnine poisoning it has been found helpful to control convulsions and cause relaxation. In producing sedation in nervous and convulsive animals, or in vicious animals to enable examination, one-half of the anesthetic dose in capsule form may be used.

Amytal is chemically isoamylethylmalonylurea and has the formula (C_5H_{11}) (C_2H_5) C.CONH.CONH.CO. It is a white crystalline powder, with slightly bitter taste, very slightly soluble in water, completely so in alcohol and ether. It is acid to litmus in aqueous solutions. The action resembles that of barbital. It must be used cautiously and may induce restlessness and excitement. The sodium salt (sodium amytal or sodium isoamylethylbarbiturate) is a white, friable, hygroscopic powder, slightly bitter in taste, very soluble in water, freely so in alcohol and insoluble in ether. It resembles amytal in action.

Dial is chemically diallylbarbituric acid or diallylmalonylurea — $(C_3H_5)_2C{\cdot}CONH{\cdot}CONH{\cdot}CO$. The drug occurs as a fine white powder, has a slightly bitter taste, is very slightly soluble in water, and completely so in alcohol and ether. It is acid to litmus in solution. It is similar to barbital in action, but is more active, and therefore should be given in smaller doses. Therapeutic doses exert no injurious effects on respiration or circulation. Hypnotic action occurs in from one-half to one hour.

Ipral is chemically ethylisopropylmalonylurea or ethylisopropylbarbiturate. The calcium or sodium salts of this drug are mainly employed. They have the structure: $Ca(C_2H_5)(C_3H_7)C{\cdot}CONH{\cdot}CO{:}NCO)_2.3H_2O$ and $Na(C_2H_5)(C_3H_7)C{\cdot}CONH{\cdot}CO{:}NCO$.

The sodium salt is less stable in solution than the calcium The latter is a white, crystalline, odorless powder, with a slightly bitter taste. It is partly soluble in water and insoluble in alcohol. Its solution is alkaline in reaction. The sodium salt has similar properties, except that it is more soluble in water. Ipral has the equivalent action of barbituric acid. It is promptly absorbed once it dissolves, and it is thought to be excreted promptly, though some effects persist for 24 hours. Therapeutic doses affect the higher cerebral centers, the heart and circulation to a negligible extent. It is said to combat restlessness and irritability, and tolerance for the drug does not seem to develop.

Neonal is chemically n-butylethylbarbituric acid or n-butylethylmalonylurea (C_4H_9) (C_2H_5)C.CONH.CONH.CO. It consists of a white, crystalline, odorless powder, with slightly bitter taste, very slightly soluble in water, readily so in alcohol and also in ether. It is acid to litmus in solution, and stable in air. The action and uses of neonal are essentially the same as those of barbital, but it is about three times as active and must be used in proportionately smaller doses. It is said to be extremely powerful in its sedative effects and is recommended for highly nervous conditions. An almost natural relaxation and feeling of comfort is claimed for this drug. Tolerance is not easily established for it. It has been found useful in persistent coughing and similar conditions.

Seconal is chemically sodium propylmethylcarbinylallylbarbiturate. This is a recently synthesized drug of the "short-acting" type. The fact that it is safer than barbital or amytal, or even sodium-pentobarbital, has given this drug a place among useful sedatives. It must be noted, however, that its effect is quite transitory.

Phanodorn (cyclobarbital) is chemically cylcohexenyl ethylbarbituric acid: $(CH_2)_4 CH:C(C_2H_5)C.CONH.CONH.CO$. It is a white, crystalline, odorless powder with a bitter taste; slightly soluble in water, readily so in alcohol and ether; and acid to litmus in solution. Its action and uses are similar to those of barbital, but it is eliminated much more rapidly, and the action is not as long-lasting as that of barbital. This makes it useful only for lighter cases of nervous irritability and perhaps for pre-anesthetic purposes.

Alurate is chemically allylisopropylbarbituric acid: $(C_3H_5)(C_3H_7)C{\cdot}CONH{\cdot}CONH{\cdot}CO$. It is a fine, white, crystalline powder, slightly bitter in taste; slightly soluble in water, completely so in alcohol, chloroform and ether. It is acid to litmus in solution. Similar to barbital in action and uses, alurate is more active and is used in correspondingly smaller dosage. Therapeutic doses are said not to affect the heart, circulation or kidneys.

Sodium alurate is the water-soluble form of alurate. It is intended for oral and rectal administration.

Nostal is chemically isopropyl-bromallylbarbituric acid $(C_3H_7 (CH_2CBr:CH_2) C.CONH.CONH.CO$. It is a colorless, crystalline, odorless powder; having a slightly bitter taste; sparingly soluble in water, ether, and chloroform; readily so in alcohol. It is acid to litmus in solution. This drug is similar to barbital, except that it is more active and therefore smaller doses are required. It is said not to affect the heart, circulation, or kidneys in ordinary doses. It is recommended to be given orally in hot drinks.

Ortal-Sodium is chemically sodium n-hexylethylbarbituric acid. $Na(CH_3CH_2CH_2{-}CH_2CH_2CH_2)(C_2H_5)$ C.CONH.CO:NCO. It is an odorless, white or yellowish powder, bitter in taste, very soluble in water and alcohol; alkaline to litmus in solution. In action and use it is similar to barbital, but is more active, and thus must be given in smaller doses.

Sandoptal: This is chemically isobutylallylbarbituric acid $(C_4H_9)(C_3H_5)$C.CONH. CONH.CO. It is a white, crystalline, odorless powder, slightly bitter; slightly soluble in water; soluble in alcohol, ether and chloroform; acid to litmus in solution. The action and use of this drug is practically the same as that of barbital.

Pentothal Sodium is a new sulphur-containing barbituric acid compound for intravenous use. It is suggested for operative procedures of short duration and has many advantages in minor surgery, dentistry, physical examination and x-ray work. Animals can be completely anesthetized with a single intravenous dose. Since the period of ataxia is short, the patient will be able to leave the hospital or office with little delay.

Allonal (allylisopropylbarbituric acid) occurs on the market as a mixture with amidopyrine. It is similar to alurate in properties, except that the amidopyrine dynamizes its action, making it powerfully analgesic.

Dose Table

(Not completely determined for all drugs or for all animals)

	Apoth.	*Metric*
Barbital and sodium barbital: (Veronal)	Dogs gr. iiss-xv	.15 -1.
	Cats gr. iss-v	.1 - .3
Phenobarbital: luminal and sodium luminal (phenobarbital sodium)	Dogs gr. ss-v	.003 - .3
	Cats gr. ¼-iiss	.0015- .15
Nembutal or pentobarbital sodium, orally	Dogs gr. iss-iii	.1 - .2
Intravenously or intraperitoneally		
Small animals; 1/5 grain per pound live weight; but not to exceed 10 grains.		
Large animals; intravenously 15-35 grains		
Ipral, ipral calcium, ipral sodium	Dogs gr. iv	.12 - .25
	Cats gr. i-ii	.006- .12
Amytal and sodium amytal	Dogs gr. ⅓-v	
Ortal sodium	Dogs gr. ii-v	.12 - .25
Nostal	Dogs gr. ¾-1½	.05 - .1

Toxicology

Although qualitatively similar, quantitatively the barbiturates differ considerably. This is indicated in the accompanying table. Though originally intended to induce sleep and rest, certain animals become nauseated and even excitable under their influence. Barbital is excreted through the urine; over half usually unchanged. Since excretion is very slow, cumulative effects appear and constitute a menace if the animal is maintained on continuous doses for any length of time. Tolerance is established in some cases and this adds greatly to the danger if the drug is pushed to obtain sedation.

The toxic effects of barbital are found especially in the blood vessels, where the action is somewhat like that of arsenical poisoning. Acute poisoning is indicated by lowered blood pressure, weakened heart action, slow and shallow respirations, depressed reflexes, temperature usually high or subnormal, cyanosis, and also asphyxia. There may be signs of renal irritation, the urine being partially or wholly suppressed. Death is often due to respiratory failure.

Autopsy findings are variable, but there is usually hyperemia, especially of the brain and liver; the kidneys may show a tubular degeneration, and edema of the lungs is sometimes observed.

With phenobarbital (luminal) the chances for poisoning are greater, since it is much more powerful in action, and the zone between the therapeutic dose and a toxic dose is much narrower. The toxic symptoms are much the same as those of barbital.

Treatment in poisoning by the barbiturates consists in pushing fluids (diuretics) to avoid renal degeneration and also to expel the drug from the system. Saline purgatives are also recommended, since some of the drug is excreted through the feces. The failing circulation (always a prominent symp-

TABLE I.

CLINICAL AND TOXIC DOSES OF BARBITURATES IN GRAMS, FOR HUMANS

(From Sollmann)

Compound	Clinical Dose	Severe Intoxication	Generally Fatal
Allonal	.3	5 -10.	10-15.
Amytal	.1-.3	1.5- 2.	2- 3.
Barbital (veronal)	.5	3 -10.	5-20.
Dial	.1-.3	2 - 2.5	2.5 plus
Pentobarbital	.1-.2	.5 plus	1. plus
Pernoston	.2-.5	.5- 1.	1. plus
Phanodorn	.1-.3	1.2	10. plus
Phenobarbital (Luminal)	.1-.2	4 - 5.	5- 9.

TABLE II.

SPEED AND DURATION OF ACTION WITH MINIMAL ANESTHETIC DOSES

(Eli Lilly & Company)

Drug	In rats intraperitoneally				Dogs orally				
	Onset of Anesthesia	Duration of Anesthesia		Complete Disappear. of Hypnosis		Onset of Anes.	Duration of Anesthesia		Compl. Disapp. of Hypn.	
	Minutes	hrs.	min.	hrs.	min.	min.	hrs.	min.	hrs.	min.
Seconal	10		30	2	30	30	2	7	12	24
Pentobarbital sodium	14		45	3	20	35	2		15	
Sodium amytal	15	1		3	50	35	2	29	22	20
Sodium barbital	35	6	40	15	50	50	6	20	48	
Phenobarbital sodium	45	8		19	20	65	7	30	60	

tom) should be counteracted by means of drugs such as adrenalin or ephedrine. More recently physiological antidotes, such as strychnine (as a spinal stimulant), picrotoxin (as a medullary stimulant), and metrazol (as a cortical stimulant) have been suggested, to be used alone or in combination, intravenously.

To illustrate the difference in toxicity of the barbiturates, cats given 25% of the fatal dose of barbital on successive days, die on the fifth day, having received 125% of the fatal dose. This indicates the cumulative nature of the compound. Using amytal similarly, giving 25% of the fatal dose for seven consecutive days, the animals appear perfectly normal at the end of this period. This indicates they must eliminate almost the entire daily dose. It shows the ease with which amytal must be destroyed in the body.

Amytal is prompt in action but its effect is of short duration; barbital has the opposite effect, being slow in onset and much longer lasting.

TABLE III.

MINIMAL ANESTHETIC AND MINIMAL FATAL DOSES

(Eli Lilly & Company)

	In rats intraperitoneally			Dogs orally		
	Minimal Anesthetic Dose	Minimal Fatal Dose	Therapeutic Index	Min. Anes. Dose	Min. Fatal Dose	Therap. Index
Seconal	mg. per kg. 40	mg. per kg. 110	2.75	mg/kg 35- 40	mg/kg 90	2.25
Pentobarbital sodium	50	110	2.20	40- 50	85	1.70
Sodium amytal	80	180	2.25	70	125	1.79
Sodium barbital	340	480	1.41	200	300	1.50
Phenobarbital sodium	150	250	1.67	100	150	1.50

THE HISTORY of medical therapeutics is literally choked with hundreds of suggestions that have appeared in standard textbooks, in medical journals, and that even were taught in our medical schools, but which today are absolutely obsolete, and deservedly so. If medicine ever becomes, not only a fine art, but also an exact science (which is the dream of every loyal and thinking physician), it will not be due alone to the labors of the research laboratories, nor to experimental studies, nor to the work of pharmaceutical chemists. Our profession will not take its place as a recognized member of the acknowledged sciences until its teachings are fortified and strengthened by actual clinical experiences of men of unquestioned integrity who are devoted to the welfare of their profession above everything else. Hippocrates gave a piece of sound advice, which is as true today as it was nearly 2,400 years ago when uttered by the "Father of Medicine": "It is Nature that heals; all that the physician can do is to favor her." This contains an incontrovertible truth which every physician would do well to remember at every bed-side to which he is called. We should pay attention to Nature, to her normal activities and defensive methods. By following them and by removing every factor that interferes with Nature's responses, we would afford her a better opportunity to heal our diseases. Surely every physician will agree that it is his bounden duty to "preserve human life, at its longest and its best." But how may this be done? By the employment of every agency that gives demonstrable aid, regardless of its origin or authorship. Such agencies are built upon a framework of the physician's student years; of his books, and medical journals; and, more important than these, his own clinical experiences and those of his colleagues.—*Frank W. Porterfield, M.D. in Clinical Medicine & Surgery.*

Traumatic Pericarditis in a Feeder Steer

By DAVID JACOBSON,
Manhattan, Kansas,
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NOVEMBER 4, 1937, a two-year-old Hereford steer in poor condition was presented for treatment at the clinic of the Kansas State College. The history of the case comprised a progressive cachexia dating back to an attack of indigestion three weeks previously, which had not responded to treatment by a local veterinarian.

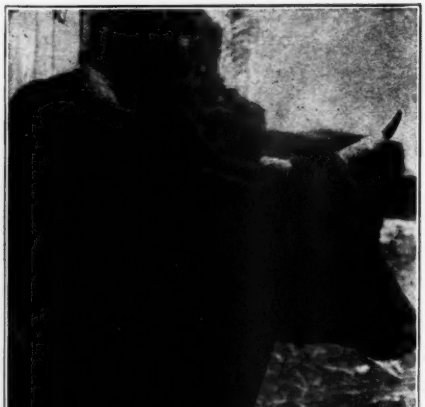
The clinical picture was as follows: temperature 104.2° F., pulse 90, respiration 40, appearance dull, head held low and somewhat extended. Slight abduction of the elbows and moderate distension of the jugular veins was evident. The hindquarters were smeary from fetid diarrhea and anorexia was complete. When forced to move the gait was stiff and labored. Auscultation of the thorax medial to the elbow revealed a characteristic rhythmic gurgle best described as the sound of water rippling over stones. The case was immediately diagnosed as traumatic pericarditis, prognosis grave.

Within 48 hours the owner gave his permission for the radical operation, which was decided upon for relief of this condition. The following operative procedure was employed:

The left side of the thorax was clipped and shaved over the area covering the fourth to the sixth ribs for a space extending about twelve inches upward from the sternum. The animal was placed on an operating table in right lateral recumbency, and the shaved area was disinfected with a 1 to 500 solution of bichloride of mercury in alcohol. The skin and subcutaneous tissues were thoroughly infiltrated with a 1.5% solution of procaine hydrochloride.

An incision about seven inches long was made with a heavy scalpel through the skin and the soft tissues directly over the center of the fifth rib extending about one and one-half inches below the costochondral articulation. With careful dissection the rib was completely isolated from all the soft structures laterally, anteriorly, medially, and posteriorly; the intercostal vessels at the posterior border of the rib being avoided by keeping close to the bone. Care was taken to leave the pleura intact. A piece of obstetrical wire saw 24 inches long was passed around the separated rib at the upper end of the incision and the bone was divided at this point. The free end of the divided rib was pulled firmly outward and twisted until disarticulation at the costochondral junction occurred.

The steer was next raised to a standing position, the left foreleg drawn forward and the exposed pleura incised. The pericardium was freely opened with the discharge of an estimated two pints of yellow, foul smelling, ichorous fluid. Exploration of the pericardial sac revealed a very sharp piece of spring steel nearly two inches long, probably a "bobby pin," which was protruding through the reticulum and diaphragm into



Typical position assumed in advanced cases of traumatic pericarditis.—*Photo by Dr. M. R. Wilson.*

the pericardium. The pericardial sac was irrigated with a very mild solution of potassium permanganate to remove accumulated pus and fibrin. The entire operative opening was left to heal as an open wound. The patient was returned to a box stall to await developments.

The Steer Described in this Report.

After-care consisted in administeration of 250cc of 50% glucose solution and 500cc of 1 to 1,000 hydrochloric acid intravenously, and thrice weekly irrigation of the pericardial sac and wound with a 1% chlorazene solution. The animal showed gradual improvement for about one week, there was a return of appetite and the body defenses were responding to the HCl treatment. At the ninth day the condition remained static and persisted thus for several days, when death occured rather suddenly; the patient having appeared somewhat bright an hour previous to being found dead. Post-mortem examination showed extensive adhesions of the pericardium, pleura, diaphragm and anterior abdominal viscera. The heart itself was roughened, the epicardium being covered with a thick fibrinous exudate.

This particular case was unusual in that traumatic pericarditis is usually found in dairy cattle which are closely confined, whereas this steer had been on the range.

TRAUMATIC PERICARDITIS

Illustration of a cow's heart with a sixteen penny nail in it. This animal gradually lost in condition for four and one-half months. She was found dead one morning, and on autopsy I found this nail penetrating the heart; one and one-fourth inches of it was still in the pericardium. Note that the surface of the heart is sloughing. The pericardial sac contained about three gallons of yellowish, offensive fluid. It is supposed that the nail was ingested with cotton seed hulls.—*R. E. Richardson*, Wichita Falls, Texas.

It is lamentably true that recoveries from the operation described in the foregoing are extremely rare, but since death is inevitable if pericarditis is not treated, the operation can be justified in certain cases where the physical condition of the patient is good and where the owner wishes that no effort be spared in attempting to save a favorite or an especially valuable animal. However, the operation should not be undertaken lightly. It is serious at best. The opening in the chest wall, of course, collapses the lung on that side and difficult and distressing breathing ensues, much to the concern of the owner.

Needless to say, indiscriminate surgery in these cases would soon put this operation into disrepute.

Abstracts

Studies of Fowl Paralysis

In an experiment in which sixty-two chickens, harboring various intestinal parasites, had been exposed *per os* to Salmonella aertrycke, all developed pathologic manifestations.[25] Five other chickens so exposed developed a complication of two manifestations. The 24 control chickens remained normal. The incubation period for fowl paralysis was 29 to 57 days; for lymphomatosis 52 to 85 days; for "light' and anemia 18 days; for myeloid leucemia 70 to 172 days; for erythroleucosis 86 to 171 days. Four complicated cases of fowl paralysis and leucemia developed in 54 to 86 days, while one case of fowl paralysis and lymphomatosis developed in 58 days.

The Study of Anemia

In every case of anemia,[26] the rate of red cell formation and delivery from the marrow, the rate of destruction, and the balance between these two factors must be determined. Measures are available for gauging accurately the state of the marrow and all important factors in red cell activity. The anemia must be studied and classified from both the clinical and laboratory standpoints.

Bee-Sting Poisoning in Dogs

Seven dogs were attacked by bees shortly after they had been chained beneath a tree.[27] When the dogs were finally released, it was found that those with black colors were stung most; next severely attacked were the roan dogs, while the white ones were left undisturbed.

Since the dogs tried to defend themselves by biting at the bees, they were mostly stung on the tongue and lips, these surfaces being almost completely covered by stings. Due to its black color, no stings could be seen on the pigmented skin.

One of the black dogs exhibited a staggering gait and rapid respiration, and convulsion at short intervals. About four hours later the swelling of the tongue was so extensive as to interfere with breathing. In another hour the dog died of asphyxia. Treatment consisted of swabbing the affected parts with ammonium chloride solution and administering spirits of ammonia aromatica internally.

Another black dog showed weakness of the hind quarters, suspension of urine and feces, and fever. He was given castor oil. As stimulant for the heart, egg-flip containing brandy was fed hourly. When the body temperature had fallen to normal, the appetite returned. The parts of the skin from which the stings were not removed, because they were invisible, became necrotic. Careful dressing and cleaning of these areas was followed by healing in two weeks.

Animal Psychology

There seems to be little agreement concerning the cause of shying. The following theories have been advanced.[28] Dexler mentions as causes the natural timidity, the ready excitability, and the low intellect of horses. Darwin rightly called shying a reflex, which would refute the latter of Dexler's suppositions. This reflex in reaction to certain stimuli sets in so quickly that there is no time left for a proper analysis of the exciting causes. Others accused changes in landscape, for it has been observed that the horse has an exceedingly keen memory, and therefore is supposed to shy when it sees objects or changes in the road which were not there when it passed previously. This theory does not offer an explanation for shying on a new road traveled for the first time. The best explanation is offered by Zell, who says: The horse is a fleeting herbivore, that is, his reaction to attacks lies in flight. Since the horse has weak eyes and, therefore, perceives objects dimly, but possesses an excellent discernment for movement, in common with all animals that see but dimly, a suspiciously swift movement will excite the flight re-

[25] Emmel, M. W. 1936. The etiology of fowl paralysis, leucemia, and allied conditions in animals. Bul. No. 305. Univ. of Florida Agric. Exp. Sta., Gainesville, Florida. Pp. 66, fig. 4, tables 18.

[26] Haden, R. L. 1937. The mechanism of anemia. *Jour. Lab. and Clin. Med.* 22:439-456.

[27] Bawa, H. S. 1937. Death of a dog after an attack by honey bees. *Ind. Vet. J.* 14:58-60.

[28] Krzywanek, — 1937. Die Tierpsychologie in der Tiermedizin. *Deutsch. Tierärztebl.* 4:166-177.

flex, and thus cause shying. Such a reaction is perfectly rational for the wild horse living on the prairie, because there are no trees or other objects capable of obstructing flight. Thus, to one familiar with the ancestral relationship of the horse this behavior cannot be credited to foolishness, in the belief that this supposition does not hold good for the domestic horse, in which running away is an irrational performance. The explanation for running into obstacles in inhabited countries lies in the fact that the fleeing horse looks backward, which feat the position of its eyes allows it to do, and consequently, cannot pay any attention to the road ahead.

This simple explanation gives also the reason the likewise dimly visioned ox, capable of defending himself, and a poor runner, does not shy, and why the likewise dimly visioned dog, an animal of prey, reacts to a foreign moving object not by flight, but by attack in the hope of finding prey.

It is a superstitious belief that the horse sees everything magnified, and therefore may under certain circumstances be terrified by an otherwise harmless object. Although the retina of the horse is indeed larger than that of man, yet the assumption is foolish. The size of the picture upon the retina can have no influence upon the size of the vision, because the process of seeing does not take place upon the retina, but in the cerebral cortex. Insofar as this should offer an explanation for shying, the object in question would have to be seen magnified in relation to its environment, which is an absurdity.

* * * *

Diagnosis of Bang's Disease

In an experiment [29] in which one cow was infected with living cultures of *Brucella abortus* and another with killed organisms of the same culture, both cows reacted positively to the conjunctival test.

In six herds, comprising 228 cattle, 26 reacted positively, and 14 reacted doubtfully to the agglutination test, while 39 reacted positively and seven doubtfully to the conjunctival test. Of those which had reacted to the serological test, seven reacted negatively to the conjunctival test.

The authors consider the van der Hoeden method specific for abortion. The disadvantage of this method is the rapid disappearance of the conjunctival reaction.

[29] Okljesa, B. and Tomasec, I. 1937. The van der Hoeden method of conjunctival allergic reaction in Bang infection of cattle (transl. title). *Veterinarski Arhiv* 7:317-328.

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Prevention of Coccidiosis of Chickens

Feeding ordinary flowers of sulphur in the ration of growing chicks was found by Herrick and Holmes [31] to prevent losses from coccidiosis. Sulphur, however, is not a cure, but a preventive. Consequently, to be of any value it must be fed at least four to six days before ingestion of the ripe oocysts.

The feeding of 2% sulphur for one week before chicks are exposed to contaminated range, and for several weeks after exposure, may therefore be found practical on farms where it is impossible to rely on strict sanitation. After the chicks have been on the range for a few weeks, and thus allowed to pick up some infection tending to develop a natural immunity, the sulphur feeding may be reduced gradually. Indications are that long-continued feeding of sulphur has some tendency to bring about rickets.

As might be expected, the feeding of considerable indigestible sulphur has a slight tendency to slow up growth, and in some cases has caused irritation in the digestive system of chicks. The hazards of feeding sulphur in considerable amounts include, in addition to the possibility of digestive disturbances and interference with growth and assimilation of vitamin D, some likelihood that a few of the chicks will get their eyelids stuck with sulphur dust so they cannot see to eat, unless their eyes are washed.

That chicks fed no sulphur will make slightly faster gains than those given sulphur is true only so long as the chicks are kept free from coccidiosis. Trials have repeatedly shown that sulphur-fed chicks will continue to thrive and grow, with fewer death losses, after infection with the disease.

[31] Bulletin No. 438, Wisconsin Agric. Exp. Sta.

On the other hand, a high proportion of those given no sulphur usually die after they have been infected with coccidiosis, and the survivors may be severely stunted in growth.

Feeding sulphur after the chicks are infected with coccidia not only fails to cure the disease, but also fails to prevent further development of the oocysts. This means that feeding sulphur to sick chickens will not make their droppings any less dangerous as a source of infection for healthy birds.

Debeaking of Chickens

The present method of debeaking [30] chickens to put an end to vices was discovered by the Ohio Station in the spring of 1928. During the past eight years the various procedures and devices, including colored ointments, colored glass, and vent or beak shields, have been tried, but debeaking has proved the most satisfactory method of control.

There is only one way to debeak properly and effectively. Only the upper beak is removed. The beak is not cut off; it is torn off. By cutting into the edge of the beak about 1-16 to ⅛ inch, ⅛ to ¼ inch back of the tip and lifting the flat side of the knife blade against the anterior portion of the beak, it is easily torn loose and removed with comparatively little discomfort to the bird and practically no loss of blood. The small cut through the one edge of the beak at the start serves only to get a hold to begin the tearing process. After the beak is torn over to the other edge, it should be pulled toward the lower beak to remove the edge of the beak. This gives the removed tip a V shape, which prevents the bird from grasping feathers or flesh and renders it harmless for two or three weeks—until the beak grows back again. By that time most birds will have forgotten their previous vices. The debeaking procedure applies to all chickens over four weeks of age. Debeaking does not prevent the bird from eating, nor does it seem to affect egg production more than handling the birds for some other reason. Debeaking is also an effective means of preventing male birds from fighting. Should certain birds become troublesome later, they may be debeaked again.

[30] Kennard, D. C. 1937. Chicken vices. *The Bimonthly Bul.* vol. 22, No. 184. Ohio Agric. Exp. Sta.

Prevention of Perosis (Slipped Tendon)

Slipped tendon [32] has for some years been prevalent where chicks are raised in battery brooders. The first noticeable symptom of the disorder is usually an enlargement of the hock joint. When the chicks are from two to ten weeks of age the tendons may slip off the bone to one side or the other. Some of the chicks recover completely, but others become permanently afflicted with flattened hocks and bowed legs. The trouble makes feeding difficult and slows up growth. In the light of the best information at hand today, it appears that the practical way of avoiding trouble with slipped tendon in chickens is to observe these precautions: Hold down the inorganic phosphorus content of chick rations by feeding only as much bone meal as is necessary. This is important, because too much inorganic phosphorus tends to bring about the slipped tendon condition. Include in the ration some of the feedstuffs that counteract the effects of phosphorus and thus help to ward off the slipped tendon condition. It has been reported that oat hulls, rice bran, and wheat shorts have this quality.

Thyroid Tumors in Dogs

Tumors of the thyroid in dogs are of multiple origin.[34] Their diagnosis is not easy for the clinician. Whether treatment should be medicinal or surgical depends upon the nature of the tumor. There are benign thyroid tumors (adenomata or simple goiter), of which the volume is variable. They are always mobile, and develop slowly, sometimes ending in malignancy. Iodine is generally efficacious in treating goiters, although certain rare cases are accompanied with toxic phenomena. The author describes one case of exophthalmic goiter (Basedow's disease).

[32] Bulletin No. 438, Wisconsin Agric. Exp. Sta.

[34] Flancette, Thése, Lyon, 1937. Abs. *Recueil de Médecine Vétérinaire*, 113:7, July, 1937.

When thyroid tumors are uniquely unilateral, thyroidectomy brings good results. Cancerous tumors cannot be distinguished from the others at the beginning, and therefore all goiters of rapid evolution should be extirpated promptly. After cachexia and metastases have developed, it is too late.

Pregnancy Disease of Ewes

Under the name of toxemia of gestation the author[33] outlines the history of the disease in various countries. It has not been noted in France, but it seems to have been recognized in the departments of Manche and Maine-et-Loire. The symptoms appear toward the end of gestation in ewes carrying twins. They consist of coma, paralysis and sometimes convulsions. Mortality is 90%. Death occurs in from three to 10 days. The 10% which survive parturition remain cachectic, and the newborn are runty, if they live. The blood shows acidosis, and the principal postmortem finding is fatty degeneration of the liver. It is generally agreed that the trouble is of alimentary origin, with consecutive troubles of lipide or glucide metabolism. Because of ignorance of its cause, there is no known treatment or prevention of value.

Comment: Except that a higher protein diet and exercise are set down as preventive, the above opinion coincides with those of H. S. Cameron and of W. L. Andrews, expressed in the Special Issue of Veterinary Medicine, on the diseases and treatment of sheep, June, 1937.

Live Culture Abortion Vaccination

From observations made in 1936, regarding the efficacy of premunition against Bang's disease in cows, the author[34] reports: (1) Of 228 pregnant cows affected with Bang's disease and treated with killed cultures (bacterins), 72 aborted; (2) of 737 non-pregnant cows affected with the disease and premunized with living vaccine two months before being bred, the percentage of abortion was 2.5% to 3.2%. Among these latter the incidence of sterility was 12%; (3) on 44 farms where living vaccine was used, no case of undulant fever was observed among persons contacting the treated cows.

Bang's Disease as a Cause of Vertebral Necrosis in Swine

In a large swine breeding establishment sundry cases of necrosis of the lumbar vertebrae were observed.[35] In three cases the author isolated lesions of *Brucella suis* organisms which showed extreme virulence for guinea pigs. A serological test of the herds disclosed that 59.5% were affected. The author attributed the osseous localizations of brucellosis to a predisposing mineral deficiency.

Vitamins in the Diet Prevent Distemper-Like Symptoms from Nutritional Deficiency in Dogs

Much of the sickness and death among dogs commonly attributed to "distemper" and other germ diseases, could actually be the result of nutritive vitamin deficiency, and as such can be prevented by proper protective diets.

This conclusion is inescapable after study of results of feeding experiments on more than 500 puppies and dogs, described in a series of articles in VETERINARY MEDICINE.

Absence or deficiency of just one vitamin factor will cause symptoms of any of the various deficiency diseases, the experiments showed. The term "distemper" is used by veterinarians and dog owners too broadly in describing a sick dog or puppy, is the belief of W. O. Frohring, author of the article—it is too convenient and general an explanation for dog illness and death.

Vitamins and nutrition in general play an important part in the general resistance of animals to infections on the one hand, and many mild and acute cases of vitamin deficiency, uncomplicated by infections, may improperly be diagnosed as distemper on the other.

[33] Royer, Thése, Lyon, 1937. Abs. *Recueil de Médecine Vétérinaire*, 113:7, July, 1937.

[34] Amiot. Efficacité et innocuité de l'emploi des vaccins vivant dans la prémunition des bovides contre la brucellose. Abs. *Revue de Pathologie Comparée*, 37:489, June, 1937.

[35] Saxer. *Jahr. fur Vet. Medez*, abstracted in Annales de *Médecine Vétérinaire*, 82:6,

For instance, in one experiment a group of puppies was deprived of all factors of the vitamin G complex. They lost their appetites, their weight fell off, and their body temperatures dropped. In some instances severe nervous symptoms developed which might easily be diagnosed incorrectly as "nervous distemper". Most of the puppies thus deprived of the several vitamin factors died within 11 days to one month.

In another series of tests, puppies were deprived of vitamin A. They quickly developed symptoms resembling "respiratory distemper" while others, protected with abundant vitamin B in their food, showed outstanding resistance, even when they were exposed in a hospital distemper ward.

Most authorities are now agreed that blacktongue is a pure nutritional deficiency disease analogous to pellagra in humans. In tests, adult dogs deprived of vitamin-B complex factors developed blacktongue and also symptoms resembling so-called intestinal distemper. Dogs on diets containing adequate amounts of vitamin B factors did not show symptoms of either disorder. When the same experiment was made on growing puppies, the young dogs deprived of vitamin-B factors died before they had any opportunity to develop disease symptoms, while control puppies receiving complete vitamin rations thrived and grew well.

The vitamin B complex, as the water-soluble series of vitamins is called, is well-named—it is indeed "complex" in that it consists of several factors, each necessary to the other and each an indispensable link in the entire vitamin chain. It is not enough that just one of the vitamin links be present —they must all be there in adequate amounts, or deficiency symptoms develop.

The vitamin B complex is further divided into groups of factors that are damaged by heat and factors that are not damaged by heat. The heat-stable factors are particularly abundant, for instance, in the watery portion of milk. One of them, the growth vitamin substance, lactoflavin, now may be extracted from milk in concentrated form and added to foods as a protection against deficiencies. In this concentration other heat-stable factors are brought down at the same time.

The interdependance of the vitamin factors is well demonstrated in a series of tests in which various single B factors were removed from, or added to, the diets.

In one test a diet complete in all necessary elements except the several vitamin-B complex factors was fed. Growth halted, weight was lost, and body temperature dropped. Lactoflavin, the growth vitamin, was added, along with a related heat-stable factor, the "P-P" factor which prevents pellagra in humans and blacktongue in dogs. Promptly growth was resumed and weight increased. This healthy response continued for several days.

However, one factor was still missing and after a time loss of weight and lowered temperature followed. At this point the missing factor—B_6—was added to the diet. The growth and weight curve went up and the temperature returned to normal.

This test, as well as many others, demonstrated the interdependent nature of the vitamin B complex—every link in the chain, B_6, B_1, "P-P" factor, lactoflavin, and other *must* be present to protect against deficiency symptoms. No extra abundance of one vitamin factor can make up for the absence or deficiency of another, any more than one especially strong link in a steel chain will compensate for a weak or broken link. However, one important fact stands out:

In the case of puppies that first were given a diet with all vitamin-B complex factors supplied liberally over a period so that their tissues were "saturated" to their storage capacity, the onset of deficiency symptoms, when the B complex was removed from the diet, was delayed. In other words, it is possible by feeding high vitamin content diets, to establish a reserve margin of general health that will carry animals through a brief period when vitamins are deficient in the diet.

The ideal means of avoiding nutritional disaster is consistent, day-after-day feeding of rations adequate in vitamins. The prevention of vitamin deficiencies is far easier than their cure.

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